Ontario Task Force
on Health and Safety
in Agriculture

Groupe d'étude de l'Ontario
sur la santé et la sécurité
dans l'agriculture

Background
Paper Number 3

Agricultural Chemicals and Farm Health and Safety

A joint project of the Ministries
of Agriculture and Food and Labour

Un projet commun du Ministère
de l'Agriculture et l'Alimentation
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AGRICULTURAL CHEMICALS AND FARM HEALTH AND SAFETY

A BACKGROUND PAPER PREPARED
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EXECUTIVE SUMMARY

Pesticides and related chemicals are highly regulated in Canada and Ontario. The sale, use, and in some instances the consequences of use, are controlled by both Federal and Provincial legislation. All pesticides intended for use in Canada must first be registered under the federal Pest Control Products Act which requires that they be subjected to a detailed toxicological evaluation. The province of Ontario further classifies pesticides into one of six schedules based upon their toxicity to man and the environment, pattern of use and persistence in the environment. The current regulations governing the availability of pesticides to agriculture in Canada and Ontario appear to be satisfactory. Dusts, particulate matter, mycotoxins, bacterial toxins and gases such as methane were identified as being significant hazards to farm workers. That no regulations or acceptable exposure guidelines exist for these substances in the farm workplace is a matter of concern. Such information is necessary in the development of strategies to limit exposure to these substances. It is noted that there is an apparent lack of awareness of these hazards and a lack of use of already available respiratory protective equipment. Little attention appears to have been given to the design of structures and equipment that would minimize exposure to these hazards.

Approximately 8.7 million kg of pesticides were used by farmers in Ontario in 1983. As a consequence of normal farming operations, it is inevitable that farm workers will be exposed to pesticides. Exposure may occur during the transport, storage, application and disposal of pesticides as well as during re-entry into fields or through contact with farm produce. While precautionary labelling, protective devices and other techniques such as the use of less hazardous formulations are available to reduce exposure to pesticides, excessive exposure does occur through misuse and negligence. The consequences of misuse of pesticides present a serious hazard to farm workers which can be significantly reduced if directions for use are followed.

Many of the safety procedures mentioned herein are already the subject of pesticide regulations which are difficult, if not impossible, to enforce at the farm level. Those farmers who have had personal experiences involving pesticide poisonings are much more likely to adhere to proper safety practices.

The evidence in the literature indicates that greatest exposure of farm personnel to pesticides occurs during the mixing/loading operation. Accordingly methods to reduce exposure via this pathway would be a priority of improved safety practices. It is also noted that inadequate attention is given to:

Compliance with existing Ontario regulations with regard to the transportation, storage, mixing, application and disposal of pesticides.

The availability, knowledge and awareness of all practical measures to avoid contact with pesticides.

The use of currently available and recommended personal protection equipment.

Increasing awareness of closed-loading systems, particularly for use with formulations of pesticides which are in the form of dusts or of powders.

The use and availability of adequate methods for clean-up of pesticide spills at the mixing/loading site and the use and availability of facilities for maintenance of proper cleanliness of person and clothing.

The formulation in which a pesticide is manufactured may have a significant effect on contact, exposure and resultant toxic effects. The use of soluble granular rather than wettable powder formulations of pesticides would help reduce concern in this area.

The toxic effects of pesticides to farm workers are usually expressed either as an acute (immediate response to the compound) or as a chronic effect (long term). A number of pesticides show high acute toxicity and, even at low doses, cause easily recognizable effects from which rapid recovery usually occurs if the source of exposure is removed. These compounds, in general, are insecticides.

The organosphosphorus insecticides such as parathion, phorate, demeton, azinphos-methyl, disulfoton and terbufos all have high to moderate toxicity and, unless used in a careful and informed manner, may be hazardous by virtue of their acute effects. For the most part they have minimal long-term effects unless exposure is continuous. Continuous exposure is unlikely to occur in normal farming operations but may result from improper ventilation of pesticide storage areas.

Some of the carbamate insecticides also have high acute toxicity and possess a hazard profile which is similar to that of the organophosphorus compounds. Carbofuran, methomyl and aldicarb all have high acute toxicity and are hazardous if not used with appropriate precautions. Carbaryl is less acutely toxic. Chronic effects would not be expected to occur with carbamate insecticides under normal use conditions.

Certain pesticides, other than insecticides, may show high acute toxicity. For example, the highly toxic herbicides paraquat, diquat and dinoseb have a mechanism of toxicity which is common to both plants and animals. These compounds are, as in the case of insecticides, only hazardous if they are not handled in the correct manner.

Many other pesticides including certain insecticides, herbicides, fungicides and nematocides can be tolerated at quite high dose with little in the way of distinct symptoms of toxicity. This tends to breed a more casual attitude to their use and handling and is of concern in the case of those compounds which may have chronic effects.

Some of these materials such as captan, alachlor, 1,3-dichloropropane and linuron have been shown to cause cancer in laboratory animals. In these laboratory tests, the pesticides are usually given to animals in high doses on a daily basis for most of their lifespan which is not typical of the pattern of farm worker exposure. The significance of cancer induced in animals under laboratory test conditions to the health of farm workers is unclear. These compounds are under active review in the U.S.A. and Canada and, until more information is available, they should be treated with the care and caution normally associated with compounds of higher toxicity.

A problem in the evaluation of the toxicity of any substance, including pesticides, is that many of the studies have to be carried out in laboratory animals. This raises the inevitable question of the relevance of observations made in animals which may not have an exactly similar physiology or biochemistry to that of man. A lack of toxicological procedures which better address the hazard of pesticides to Ontario farm workers is noted. The published information on both fatal and non-fatal poisoning in Ontario farmers is inadequate in that it does not provide a comprehensive analysis of the incidence, geographic location, occupation and severity of pesticide poisoning in Ontario. Chemicals other than pesticides, such as silo gases, have produced fatal poisonings in Ontario, however, these occurrences have been rare and no firm data on the effects of these chemicals exist for Ontario farm workers.

While epidemiological studies, conducted primarily in other countries, have suggested a link between the occupation of farming and the development of certain types of cancer, there is no data linking specific pesticides or other farm chemicals with increased cancer rates in farm workers. It is noted that there is a lack of epidemiological studies which address the effects of pesticide use on the health of farmers in Ontario.

While much information on safe handling of pesticides and other chemicals is available from a number of sources, it appears to be under-utilized by Ontario farmers. While only limited data exist for Ontario, observations from other provinces suggest this problem is widespread in Canada. This may be due, in part, to inadequate presentation and distribution of safety information on pesticide products. In addition, dealers and agricultural representatives supply much of the safety information on pesticides to the farm community. It is noted that these persons may lack formal training in the hazards associated with pesticide use. In a similar vein, the lack of adequate training and/or certification programs for farmers and agricultural workers in the safe use of pesticides was noted.

Little information on the hazards associated with chemicals other than pesticides is being made available to Ontario farmers and farm workers.

CHAPTER ONE

INTRODUCTION

Terms of Reference

The Ontario Task Force on Health and Safety in Agriculture was established in 1983 as a joint project of the Ministries of Agriculture and Food and of Labour. The Task Force was instructed to determine the need for protection of the occupational health and safety of farm workers and their families. The specific terms of reference are as follows:

1. The Task Force is established by the Minister of Agriculture and Food and the Minister of Labour of the Province of Ontario and its members will be appointed by documents signed by both Ministers.

2. The Task Force is constituted to carry out the task defined in these terms of reference and will cease to exist when that task is completed.

3. The function of the Task Force will be to investigate and report on the need for protection of the health and safety of farmers, farm workers and members of farm families engaged in farm work. Among the matters the Task Force will consider are:

   - the nature of occupational health and safety hazards in agriculture;

   - where the need for protection exists, that is, what occupations, farm work activities and types of farming;

- how the occupational health and safety experiences of persons engaged in on-farm work vary by age, form of attachment to the industry, length of service and other relevant personal variables;

- the problems of defining a farming operation and a farm workplace;

- mechanisms for providing protection against health and safety hazards in farm work; and,

- if the conclusion is that legislation is required, the areas to be addressed.

4. The Task Force will present its findings to the Ministers of Agriculture and Food and of Labour, in a fully documented final report and, as deemed appropriate, interim reports will be made to a joint steering committee of officials drawn from both Ministries.

Within this larger framework, the Canadian Centre for Toxicology was assigned the task of developing a review paper which would describe and evaluate the health and safety hazards posed to both users (farmers and farm workers) and bystanders by the use of agricultural chemicals and the generation of agricultural chemicals on the farm. In particular, the study was expected to:

1. Identify and list the agricultural chemicals used in Ontario, excluding fertilizers. The chemicals will be ranked in terms of volumes used and known degree of toxicity.

2. Identify and rank chemicals generated on the farm.

3. Classify the chemicals in conformance with the system used by the Ontario Ministry of the Environment to classify pesticides.

4. Describe and assess each major group of chemicals with regard to toxicology, the nature and extent of exposure and the associated risks, and the findings of related epidemiological work.

5. Select and discuss in detail those chemicals deemed most likely to cause injury or illness.

6. Identify and rank the major hazards arising from the use and generation of agricultural chemicals. Where possible, protective measures will be suggested.

Background

Farming, like any occupation, poses a number of health risks to those who engage in it. Some of these risks are comparable to other occupations while others are quite different, such as the risks arising from the fact that the farm worker and his family often live right in the "workplace". This report looks at those risks generated by the use of or exposure to a broad range of agricultural chemicals. There are basically two general groups of chemicals to which farm workers are exposed, those chemicals which are brought on to the farm and those which are generated on the farm as a result of farming operations. The former group is largely composed of pesticides of various types, which have been used increasingly over the last few decades to enhance agricultural efficiency through the control of losses from weeds and pathogens. The pesticides include herbicides, insecticides, fungicides, fumigants, nematocides, rodenticides and repellents. In this paper, certain growth regulators are also included since they are included in the statistical data on pesticide use in Ontario.

The other group of chemicals to be discussed are those generated on the farm. Particulate matter or dust often contains an inert component such as particles of hay or soil and associated fungi or bacteria which may include toxins such as tricothecenes (e.g. vomitoxin). Dust is generated at many points in the field and in the barn. Noxious gases such as carbon dioxide are added to grain silos and other gases are generated by silo storage and decomposition of crops. Gases are also generated by animal wastes which are often stored in tanks for subsequent use as fertilizer.

A number of chemicals will not be considered in this study, among them fertilizers and petroleum products (and additives) which are in significant use on the farm but are outside the terms of reference. Because of the complex nature of the agricultural industry, individual differences in farming operations and a lack of good data, it will not be possible to identify the hazards associated with any particular type of operation or area of the province.

The major focus in the study was pesticides, since they represent a significant portion of the chemicals used on the farm and present the greatest concern for both acute and chronic effects amongst both the general population and the farming population. Little information was available on the use of pesticides in Ontario until 1973 when the Ontario Ministry of Agriculture and Food conducted the first of a series of surveys on pesticide use, to be carried out every five years, at the request of the International Joint Commission (IJC) (Roller, 1975). The IJC was particularly concerned with the contamination of fish and wildlife in the Great Lakes through runoff and other means of transport of these pesticides from the field into the lakes.

The use of pesticides has grown considerably since 1973 (Table 1-1). It is interesting to note from this table that the quantities of pesticides used have grown considerably while the areas grown have been relatively stable.

Table 1-1

Pesticide Usage In Ontario

Item	1973	1978	1983
Pesticides used on fruit and vegetables (kg)	985500	771300	918700
Area grown (ha)	—	99400	101300
Pesticide used on field crops (kg)	4508600	5802600	7800500
Area grown (ha)	3994500	4059700	4060500
Total Pesticides used (kg)	5494100	6573900	8719200
Total area grown (ha)	—	4159100	4161800

Reference: Roller, 1975; 1979, McGee, 1984.

The surveys from which Table 1-1 was drawn cover the pesticides applied to crops in Ontario but do not include the small quantities of pesticides which may be purchased for limited use around barns or other outbuildings or within the farmworker's house. It would appear to be impossible to quantify this particular usage of pesticides. The surveys by Roller and McGee also include the use of growth regulators, the use

of which is primarily on tobacco crops. The apparent drop of pesticide use in 1978 in fruits and vegetables may be due to a reduced need for fungicides and insecticides, possibly as a result of different weather conditions. Quantification of the amounts of chemicals generated by farming operations is an entirely different prospect, in that there are no good statistics available. The amount of dust or noxious gases to which the farm worker is exposed have been studied in a very few instances and not in any comprehensive manner. Exposure to some of these chemicals is bound to be increasing, as there are now 40,000 silos and 15,000 to 20,000 liquid manure pits in operation, with the numbers of each growing every year (Swinn, personal communication).

Selection of Chemicals

The time available for the study did not permit an extensive review of the over 125 pesticides which are listed in the 1983 survey by McGee. In order to effect as comprehensive a coverage as possible, most of the chemicals are discussed in broad terms by class. For example, the organophosphorus insecticides have roughly the same chemical structure, are used in the same manner, and have similar actions on target species and on humans. Such groups have been treated as a unit where it is reasonable to do so.

From the extensive list of farm chemicals to be considered, the Canadian Centre for Toxicology developed a list of approximately twenty chemicals which were of particular concern. The chemicals were ranked on the following factors:

volume of use
frequency of application
exposure potential during application
acute and chronic toxicity

carcinogenic or mutagenic potential
most common formulation
classification in the MOE schedule

The members of the study team performed this ranking independently. The results were then combined in an effort to reduce any individual bias and achieve a more objective assessment. A similar assessment by the U.S. Environmental Protection Agency (Milby, 1975) provided corroboration on the relative rankings for some of the chemicals. The preliminary list of chemicals was then reviewed with the Task Force and a number of experts in the pesticide field. Minor changes were made to arrive at the final list of chemicals in Table 1-2. These chemicals are dealt with in more detail later in the paper, in addition to the more general review of the classes of chemicals.

Table 1-2

List Of Chemicals For Detailed Study

1, 3-dichloropropene and 1,2-dichloropropane	dinoseb
alachlor	disulfoton
aldicarb	endosulfan
atrazine	linuron
azinphos-methyl	maneb
captan	MCPA
carbaryl	methomyl
carbofuran	paraquat
chlorothalonil	parathion
demeton	phorate
	terbufos
	tricothecenes

CHAPTER TWO

LEGISLATION OF FARM CHEMICALS

Pesticides

Agricultural chemicals such as pesticides, drugs and fertilizers are regulated and controlled by a large number of acts and regulations in Canada and Ontario. Pesticides are one of the most heavily regulated agricultural chemicals and are defined under the Federal Pest Control Products Act as:

> control product means any product device, organism, substance or thing that is manufactured, represented, sold or used as a means for directly or indirectly controlling, preventing, destroying, mitigating, attracting or repelling any pest, and includes
>
> (a) any compound or substance that enhances or modifies or is intended to enhance or modify the physical or chemical characteristics of a control product to which it is added, and
>
> (b) any active ingredient used for the manufacture of a control product;

The Ontario Pesticides Act and Regulations define a "pest" and a "pesticide" as follows:

"pest" means any injurious, noxious or troublesome plant or animal life other than man or plant or animal life on or in man and includes any injurious noxious or troublesome organic function of a plant or animal;

"pesticide" means any organism, substance or thing that is manufactured, represented, sold or used as a means of directly or indirectly controlling, preventing, destroying, mitigating, attracting or repelling any pest or of altering the growth, development or characteristics of any plant life that is not a pest and includes any organism, substance or thing registered under the Pest Control Products Act (Canada);

The sale, use, and in some cases the consequences of pesticide application in Canada and Ontario are regulated by the laws in Table 2-1.

Table 2-1

Laws of Major Pertinence to Pesticides

Federal Acts and Regulations
Pest Control Products Act and Regulations
Food and Drug Act and Regulations
Environmental Contaminants Act
Fisheries Act
Migratory Birds Convention Act
Ocean Dumping Control Act
Canada Water Act
Northern Inland Waters Act
Arctic Waters Pollution Prevention Act
Transportation of Dangerous Goods Act
Ontario Provincial Acts and Regulations
Pesticides Act and Regulations

Pesticides are registered in Canada under the Pest Control Products Act which is administered by Agriculture Canada in consultation with one or more sections of the following departments:

Health and Welfare Canada

Environment Canada
Fisheries and Oceans Canada

Criteria which are examined during registration under the Pest Control Products Act are listed in Table 2-2.

Re-evaluation of registered pesticides by the Control Products Section of Agriculture Canada may also be carried out from time to time as new information on the product or its active ingredient becomes available or when a manufacturer requests a new use or significant extension of use. In addition to registration of a product, pesticides are classified by the Control Products Section of Agriculture Canada into one of three classes:

Domestic, Commercial and Restricted.

Table 2-2

Canadian Pesticide Registration Requirements

1	Label
2	Product Chemistry
	2.1 Active Ingredient Specifications
	2.2 Product Identity
	2.3 Manufacturing Processes
	2.4 Specifications for Technical Materials
	2.5 Analytical Methods
	2.6 Physical and Chemical Properties
3	Toxicology
	3.1 Acute Studies - Technical

- 3.2 Acute Studies - Formulated
- 3.3 Short-Term Studies - Technical
- 3.4 Short-Term Studies - Formulated
- 3.5 Long-Term Studies
- 3.6 Special Studies

4 Metabolism Studies

5 Food, Feed and Tobacco Residue Studies

6 Environmental Chemistry

7 Environmental Toxicology
- 7.1 Birds and Mammals (Wild)
- 7.2 Aquatic Organisms
- 7.3 Non-Target Invertebrates

8 Efficacy

The Ontario Pesticides Act and Regulations require that all pesticides registered under the federal Pest Control Products Act and which are to be used in Ontario also be submitted to the Ontario Pesticides Advisory Committee for classification. As in the federal regulations, classification is based on all aspects of the toxicity of the compound to man and in the environment as well as its use pattern, degree of persistence and other factors. The purpose of such classification is to restrict access to the pesticide to only those who clearly need it and who have the training and knowledge to use it in the correct manner.

This Ontario classification system results in the compound being placed in one of six schedules (Ontario Guidelines November, 1984) which are described below.

Schedules 1 and 5

Schedule 1 pesticides are restricted and can only be used under the authority of a specific use permit. Schedule 5 pesticides are limited to application on agricultural land. Sales of both Schedule 1 and Schedule 5 pesticides are permitted only through wholesale vendors and holders of Class 1 retail vendor licences. A record must be kept of each sale.

The criteria for defining Schedule 1 include:

1) pesticides that pose a serious hazard to public health and/or the natural environment;
2) pesticides that are persistent and/or give rise to persistent metabolites that produce undesirable side effects on non-target organisms either by acute or chronic toxicity;
3) pesticides which through their mode of action may inflict unnecessary suffering to pest vertebrate animals;
4) pesticides classified as "restricted" by the Federal Pest Control Products Act.

Criterion for defining Schedule 5 is:

pesticides normally in Schedule 1 but which are essential for the protection of agricultural crops and for which suitable substitutes do not exist.

Schedule 2

Pesticides and/or pesticide formulations in this group are restricted to agriculturalists, licensed exterminators and registered custom sprayers. Sales are permitted through wholesale vendors and holders of Class 1 and Class 2 retail vendor licences. Sales records must be kept.

The criteria for defining Schedule 2 include:

1) pesticides that could pose a hazard but are considered suitable for use by the experienced professional applicators if used according to recommended procedure.

2) organic pesticides that do not present problems of long term persistence or accumulation in biological tissues, and those inorganic pesticides that may present a degree of hazard to the environment.

Schedule 3

Pesticides and/or pesticide formulations in Schedule 3 may be made available for domestic purposes if the hazards accompanying their use are considered minimal. Sale of Schedule 3 pesticide products is restricted to wholesale vendors and holders of Class 1, Class 2 or Class 3 retail vendor licences. Sales records are not required.

The criteria for defining Schedule 3 include:

1) pesticides or dispersants should pose minimal hazards to the environment or to public health if used according to recommended procedure;
2) organic pesticides that are short-lived and do not produce either persistent or toxic metabolites;
3) those inorganic pesticides that present a minimal environmental hazard;
4) product residues should not pose a problem when "empty" containers are disposed of in municipal garbage.

Schedules 4 and 6

Schedule 4

Pesticides and/or pesticide formulations in this group are those that can safely be handled by any type of outlet and would be available for sale in food handling establishments. Wholesalers are required to have at least a limited wholesale vendor licence but no vending licence is required at the retail level.

The criteria for defining this group include:

1) pesticide formulations that can be considered relatively innocuous to humans. This includes compounds that are currently available for non-pesticide uses, or are used as insect or animal repellents classified as "Domestic" under the Federal Pest Control Products Act, or are pesticides formulated in very low concentrations;
2) pesticides that are of no known hazard to the environment or to domestic pets;
3) all products must carry a federally approved "Domestic" label;
4) maximum package content must not exceed 1 kilogram by weight or 1 litre by volume, and all containers must be physically inspected and approved by the Pesticides Advisory Committee after acceptance of the active ingredients as Schedule 4 candidates.

Schedule 6

Pesticide products assigned to this group are identical to those in Schedule 4 but there is no limit to package size and the products may also be designated for commercial use. Schedule 6 pesticide

products may be sold by wholesale vendors, limited wholesale vendors and holders of Class 1, Class 2, or Class 3 retail vendor licences.

The toxicity guidelines used in classification are based on the lowest toxic doses or concentrations obtained in animals such as primates, dogs, cats, rodents, birds and fish. Carcinogenic, teratogenic and mutagenic potential are considered as well as chronic and acute lethal toxicity. Acute toxicity criteria are given below in Table 2-3.

Table 2-3

Guidelines For Oral, Dermal And Inhalation Toxicity Evaluation

	Schedules 1 and 5	Schedule 2	Schedule 3	Schedules 4 and 6
Acute Oral LD_{50} single dose-mg/kg	0-50	50-500	500-5000	over 5000
Acute Dermal LD_{50} single dose-mg/kg	0-100	100-1000	1000-10000	over 10000
Acute Inhalation LC_{50} (continuous for 8 hours-mg/L air)	0-2	2-20	20-200	over 200

Vendor licences restrict the storage, sale and display of pesticides in such a manner that pesticides may not come into contact with food or drink for human or animal consumption. In addition, the storage area must be ventilated to the outside and unauthorized access to it restricted. Additional restrictions apply to sale, display, storage and

fire department notification in the case of sale or storage of pesticides in Schedules 1, 2, 3 or 5.

Other Chemicals in the Farm Workplace

A large number of other chemical substances may be used or produced on farms and, as indicated in Chapter 4, some of these may be hazardous. No regulations currently exist for the control of dusts, mycotoxins and fungal or bacterial material or gases such as methane, hydrogen sulphide or ammonia which may be generated during agricultural operations. Regulations which do limit exposure to these chemicals apply to the industrial workplace and farms are specifically excluded from this application.

Discussion

The current regulations governing the availability of pesticides to agriculture in Canada and Ontario appear to be satisfactory. However, the lack of acceptable exposure guidelines to other hazardous substances in the farm workplace is a matter of concern. Such information is necessary in the development of strategies to limit exposure to these substances.

CHAPTER THREE

PESTICIDES

Pesticides and Their Usage in Ontario

The amounts of pesticides used on farms in Ontario in 1983 have been estimated from a survey of farmers (McGee, 1984) and are shown in Table 3-1.

Table 3-1

Estimates of Pesticides Used in Ontario in 1983

Pesticide	Type	Amount (kg)	Pesticide	Type	Amount (kg)
atrazine	h	1729680	simazine	h	3000
dichloropropenes/panes	n	1350380	difenzoquat	h	2910
alachlor	h	1060640	isofenphos	i	2710
metolachlor	h	842640	methomyl	i	2640
decyl alcohol	g	590270	methoxychlor	i	2570
cyanazine	h	431820	oxydemeton -methyl	i	2550
butylate	h	298450	methamidophos	i	2410
metribuzin	h	200120	acifluorfen	h	2400
methylisothiocyanate	n	185800	trichlorfon	i	2360
2,4-D	h	184480	diuron	h	2200
MCPA	h	157210	paraquat	h	2020
linuron	h	152090	naptalam	h	1780
metiram	f	139080	permethrin	i	1720
mancozeb	f	111410	fenvalerate	i	1710
captan	f	104260	propargite	i	1710
sulphur	f	79340	dimethoate	i	1610
trifluralin	h	79340	dichlone	f	1590
glyphosate	h	76350	diquat	h	1520
chloropicrin	n	73970	dichlofop-methyl	h	1380
terbufos	i	72340	bensulide	h	1360
chlorothalonil	f	58090	demeton	i	1290
2,4-DB	h	55380	pyrazon	h	1240
chloramben	h	47100	dalapon	h	1190
dicamba	h	45130	terbacil	h	1090
carbofuran	i	43890	metalaxyl	f	1080

Table 3-1 (Continued)

Estimates of Pesticides Used in Ontario in 1983

Pesticide	Type	Amount (kg)	Pesticide	Type	Amount (kg)
fonofos	i	40130	chinomethionat	i	1070
phosmet	i	36920	chlordane	i	1040
carbaryl	i	36480	formetanate hydrochloride	i	1010
phosalone	i	36430	improdione	f	890
azinphos-methyl	i	30480	amino triazole +	h	880
EPTC	h	30330	cypermethrin	i	820
bentazon	h	29750	dinoseb	h	790
chlorpyrifos	i	29250	dichloran	f	750
metobromuron	h	27930	fluazifop-butyl	h	610
2,4-DP	h	24600	zineb	f	590
maneb	f	22070	ethephon	g	550
MCPB	h	21540	thiram	f	540
allidochlor	h	19160	fensulfothion	i	500
captafol	f	18430	ioxynil	h	460
acephate	i	16120	methidathion	i	430
diphenamid	h	14470	chlorothal -methyl	h	420
diazinon	i	13990	naled	i	380
disulfoton	i	10640	dichlobenil	h	340
endosulfan	i	9160	mevinphos	i	280
cyhexatin	i	7810	metam-sodium	n	260
fixed copper	f	7250	napropamide	h	250
dinocap	i	7160	dazomet	n	200
bromoxynil	h	7080	pirimicarb	i	180
dodine	f	6440	fenbutatin oxide	i	150
ferbam	f	6370	chlorpropham	h	130
dicofol	i	6320	anilazine	f	120
malathion	i	6190	chloroxuron	h	120
phorate	i	5350	prometryne	h	120
aldicarb	i	5230	lindane	i	110
sethoxydim	h	4430	propyzamide	h	110
benomyl	f	4360	propanil	h	100
monolinuron	h	4340	deltamethrin	i	90
mecoprop	h	4170	chlorfenvinphos	i	80
folpet	f	4140	streptomycin sulfate	f	70
Bacillus thuringiensis	i	3930	mefluidide	h	20
parathion	i	3590	thiophanate-methyl	f	20
pebulate	h	3040			

h=herbicide i=insecticide f=fungicide n=nematocide g=growth regulator

A general observation from this data is the greater percentage of herbicides in comparison to insecticides and fungicides used in Ontario and the high use of the dichloropropane-type nematocidal soil fumigants. This reflects two factors, the increased use of herbicides in place of mechanical cultivation and the climate of the region which dictates the type of crop and thus tends to minimize the impact of insects as pests.

Pesticide Exposure and Toxicity

All members of the farming community in Ontario including farmers, farm workers, farm families and bystanders may be exposed to the pesticides used in agriculture. In general, the hazard associated with the use of pesticides is dependent on the following factors: a) pesticide levels in the work environment; b) the amount of pesticide contacting the body; c) the amount of pesticide entering the body; and d) the metabolism and toxic action of the pesticide. The amount of pesticide which reaches the site of toxic action is a function of the first three factors. The effects of pesticide poisoning may be classified as acute, delayed, chronic or allergic. In acute poisoning, sudden distinct symptoms are seen shortly after absorption of large amounts of the chemical agent. Delayed poisoning may be of various degrees of severity and may occur after a short exposure followed by a latent period before symptoms appear.

Chronic poisoning may occur after repeated exposure and absorption of relatively small amounts of pesticide over an extended period. Very little confirmed information on chronic effects of pesticides on farm workers is available, however extrapolation from illness in pesticide manufacturing workers and from experimental animal studies suggests a potential for harm in some cases. Among the effects shown in animals treated for prolonged periods or at strategic times, but with relatively large doses of certain pesticides, are neurotoxic signs, reproductive

effects, teratogenesis and organ pathology including carcinogenesis. In the following sections the toxicity and hazards associated with pesticides will be discussed in regard to pathways of exposure, routes of entry, toxicology of the pesticides and the incidence of pesticide poisoning.

Pathways of Pesticide Exposure

In this section the pathways of exposure are discussed in relation to the primary activities involved with pesticide use on the farm, i.e. transport, storage, mixing/loading and application (including worker re-entry and bystander exposure) and disposal.

Transport

Pesticides are usually transported to the farm in the rear of a truck or other farm vehicle. Small amounts of pesticide may be transported inside the passenger compartment. During transport, exposure of humans to the pesticide is unlikely unless the packaging material is damaged.

While the sale of agricultural pesticides in damaged containers is not legal, it may occur. The transport of leaking pesticide containers may result in exposure of the farmer to significant amounts of pesticide during transport, especially if personal protection equipment is not worn during loading or unloading of the vehicle.

Damage to packaging materials resulting in the leakage of pesticide may occur as a result of a vehicular accident during transport. Emergency response teams are available to deal with vehicular accidents resulting in large releases of toxic chemicals, e.g. the Transportation Emergency Assistance Plan under the direction of the Canadian Chemical Producers Association which coordinates agrochemical emergency response teams;

CANUTEC under the direction of Transport Canada; and the National Environmental Emergency Centre under the direction of Environment Canada. However, these teams are rarely involved with the relatively small-scale accidents that involve transport of pesticides to the farm. The disposal of damaged containers or the spillage of pesticide which may result from vehicular accidents is usually the responsibility of the farmer. The exposure of personnel to pesticides during the accident itself would be unlikely unless pesticides were being transported within the passenger compartment of the vehicle and the packaging material was damaged to such an extent as to cause leakage. If personal protection equipment (see mixing/loading for a detailed discussion of this equipment) is not used during the decontamination and cleanup following an accident, the farmer and other personnel at the accident site may be exposed to significant amounts of pesticides.

In relation to the total amount of pesticide transported by farmers, the above instances are believed to be rare and the exposure of farmers during transport to significant amounts of pesticide is unlikely to occur under normal conditions. The only information available on accidents during the transportation of pesticides is anecdotal and it is difficult to estimate the extent of the problem or the actual hazard involved.

Storage

Following transport to the farm, pesticides may be stored for periods of several weeks or months. In Ontario, the safe storage of pesticides on the farm is enforceable by law. Pesticides must, by regulation, be stored in areas where access is controlled at all times, the pesticide storage area must, by regulation, be vented to the outside, only pesticides and their adjuvants should be stored in the area, and a notice that the area is used for pesticide storage must, by regulation, be clearly visible on the outside of the storage area. These regulations require the storage of pesticides in locked rooms in farm buildings, or a separate

locked storage shed. The storage of pesticides in secure areas reduces the possibility of theft, reduces the potential of contamination of feed and equipment, reduces the fire hazard, and minimizes the opportunity for exposure of unauthorized personnel to the pesticides.

Unfortunately, pesticides are not always stored in a secure area, or the area may be left unlocked and unattended during the mixing/loading and application operations. The security of the pesticide storage area is especially of concern in situations where significant numbers of people unfamiliar with farm operations visit the farm, e.g. pick-your-own operations. These farms are almost invariably associated with fruit or vegetable production in which significantly greater amounts of highly toxic pesticides are used. A similar situation occurs in ornamental production where the public may be allowed to select their plants directly from the greenhouses. In addition, farm laborers or visitors to farms may take pesticides from storage areas to control pest problems in their own gardens and homes without the knowledge of preharvest intervals, safety procedures, or proper application equipment. Children playing in the storage area may be exposed to dusts and spilt pesticides. As children see adults using the pesticides, they may mimic the actions of these adults, producing their own mixtures of pesticides, or playing with the pesticide concentrates. A related problem with regard to the poisoning of children is the storage of pesticides in other containers, especially those associated with foods, i.e. pesticide stored in beverage bottles. By law, pesticides cannot be stored in any other containers than those specifically designed for that purpose and, conversely, pesticide containers may not be used for the storage of other products.

Poorly vented pesticide storage areas may result in the exposure of farmers and farm labourers to significant amounts of dust and vapours containing pesticide. Thus, as required by law, pesticide storage areas must be well ventilated. Opened pesticide containers should be resealed

as securely as possible and the storage area should be kept free of spilt pesticide.

In the event of a fire in the pesticide storage area, personnel involved in extinguishing the fire should not be exposed to the smoke and fumes produced. In addition, special precautions should be taken due to the flammable and explosive nature of some pesticides. This is the primary reason for the storage area being designated as such with a sign.

It is difficult to document hazards associated with storage of pesticides but the data presented in Tables 3-2 to 3-4 suggest that many cases of pesticide exposure and poisoning occur in young children. This is a clear indication of a lack of adequate control of pesticide storage but the data from Statistics Canada do not allow the identification of farm related pesticide poisonings from incidents in other environments.

Mixing, Loading and Application of Pesticides

The Mixing/Loading Site

The pesticide storage area may or may not be adjacent to the area where the pesticide is mixed and loaded into the application equipment. In the latter case, the pesticide should be transported to the mixing/loading location on a daily basis. This location may be in the field if the pesticide is applied in the form it is received, i.e. granular pesticides, or near a source of water if the pesticide is to be applied with a sprayer. As the mixing/loading area is usually not supervised when the application is being carried out, the temporary storage of pesticide in the field may provide access by children or other unauthorized personnel.

The mixing/loading site is often contaminated with pesticides. The concentrated pesticide may be spilt, or spillage may result from the

overflow of unattended spray tanks as they are being filled with water. Rarely are materials maintained in the mixing area for the absorption of spills. Spilt material is usually left to soak into the ground, volatilize, or run off into ditches. Spillage at or near the head of wells may result in the contamination of wells used as a source of potable water on the farm. Similarly, potable water may be contaminated if the sprayer is filled using a pump that is not equipped with an anti-siphoning device which would prevent backflow of the spray mixture. The fact that the mixing/loading area is often contaminated with pesticides may provide a direct threat to personnel due to potential contamination of clothing, food, and beverages stored or consumed in the area. It may also result in long-term exposure of all farm personnel, including the farm family, to low concentrations of pesticides in potable water.

Mixing/Loading Personnel

The potential hazard of pesticides to those involved in the mixing/loading operation is well recognized. Usually the mixing/loading operation results in greater contact with pesticides than any other operation on the farm. During the mixing/loading operation, spillage of the pesticide concentrate may occur, and personnel may continually come into contact with dusts or vapours from the pesticide.

Pesticide formulations can be ranked from those which cause the greatest risk of contact during the mixing/loading operation to those with least risk as follows:

dusts
wettable powders
emulsifiable concentrates (high and low volatility)
granulars
soluble granules

The application of pesticides formulated and applied as dusts is rare in agricultural production, because of the mixing/loading and drift hazard. However, many pesticides are formulated as wettable powders which are almost impossible to open and load into the spray tank without contact by the mixer/loader. To a lesser extent, emulsifiable concentrate formulations with high volatility may result in contact with pesticide fumes. Some pesticides are formulated so as to reduce this volatility problem, e.g. phenoxy herbicides are available in low volatility ester and the less volatile salt formulations. These commonly used formulations, wettable powders and emulsifiable concentrates, often contain relatively high concentrations of active ingredients which further increases the risk associated with the mixing/loading of these materials. Granular formulations usually have lower concentrations of active ingredients and do not produce as much dust as wettable powders. A soluble granule formulation usually involves formulating wettable powders into small granules which dissolve upon contact with water. This method of formulation substantially reduces the amount of dust released during the mixing/loading operation.

Personal protection equipment which may be worn during mixing/loading and other operations to reduce the contact with pesticides are discussed below.

Gloves

Industrial rubber gloves made from natural rubber without a cloth lining or neoprene gloves should be worn when handling pesticides. However, rather than the proper type of gloves the farmer may use those which are normally available on the farm, i.e. cloth or leather gloves. These provide little, if any, protection. Gloves may not be of sufficient length to cover the forearms with the result that pesticide may run down the gloves onto the arm. Contaminated gloves may or may not be rinsed or washed after use resulting in contact when they are handled or re-used.

Often the greatest chance of direct contact with large amounts of pesticide during the mixing/loading operation is to the hands and forearms. The importance of wearing proper gloves to minimize this contact cannot be underestimated.

Respirators

Respirators are specifically worn to reduce the inhalation of dust and toxic substances. Not all respirators are suitable for protection against pesticides, e.g. dust masks only provide significant protection from dusts. Farmers should use respirators approved by NIOSH (National Institute For Occupational Safety and Health) for protection against agricultural pesticides. Cannisters on the respirators should be replaced regularly. In general, replacement is necessary after eight hours of use or if breathing becomes difficult or any pesticide odour is noticed.

Respirators should always be worn during the mixing/loading operation, however, respirators are uncomfortable items of equipment and therefore are seldom worn. This is especially true if the farmer has facial hair. A normal agricultural respirator will not seal properly to a face with a beard or side-burns. A farmer with a beard is forced to resort to a full-face respirator which is even more uncomfortable. Some farmers will use vaseline around the edge of the respirator to try and obtain a better seal.

Coveralls/Apron

The farmer rarely has a set of work clothes used only during the pesticide mixing procedure. Work clothes, including coveralls or aprons contaminated with pesticide dusts or spills, are often worn the entire day and may even be worn for a series of days without washing. These clothes are not only a source of exposure to the farmer and farm workers, but also result in the exposure of other family members. For

example, children may play in areas where the clothes are stored (i.e. mud rooms), and thus be exposed, or laundering the clothes results in the exposure of those involved in the laundering process. Laundering may not remove all pesticides from clothing and the laundering of contaminated with non-contaminated clothing may result in pesticide residues in all of the clothing. Clothing which has been exposed to pesticides ideally should be changed or washed daily and always washed separately.

Eye Protection

Protection for the eyes is not usually worn, except by those farmers who normally wear safety eyeglasses. The eyes may be contaminated by spills, by dusts and vapours, and by the vigorous agitation in spray tanks and resultant splashing.

Boots

Normal work boots made of leather do not provide sufficient protection as they will absorb many pesticides. Rubber boots should be worn during mixing/loading or during the clean-up of any spilt pesticide.

Hats

A farmer rarely wears a hat specifically for protection against pesticide exposure during mixing/loading. Hats for protection against pesticide exposure are available, however, the only instances in which they are used to any extent is during the application of pesticides within structures or application with an airblast sprayer.

During the mixing/loading operation most of the contact dose would impinge upon the body through dermal contact and to a lesser extent by inhalation. The failure to wear protective clothing during the

mixing/loading operation is probably the most common cause of pesticide poisoning in farmers. It should be appreciated that a farmer wearing a respirator, coveralls, neoprene or rubber gloves, rubber boots, and possibly a rubber apron is not comfortably dressed. This is especially true if weather conditions are hot and humidity is high. As a result, the personal protection equipment is often not used. Some personal protection equipment may actually result in pesticide spillage as it limits the freedom of movement of the farmer and may make pesticide containers difficult to handle. However, the importance of the personal protection equipment in the use of pesticides cannot be overemphasized. In some areas of the United States, closed-loading equipment and medical supervision for all mixer/loading personnel is required if they are working with pesticides of high toxicity for more than a specified number of hours per month. Closed-loading equipment is rarely used in Ontario and medical supervision is not required.

Application

The Applicator

Once the pesticide is ready to apply, i.e. in the spray tank or pesticide hopper, it is usually in a relatively dilute form. As a result, the pesticides are much less likely to result in acute toxic effects than during the previously discussed operations. However, some pesticides are sufficiently toxic to cause significant direct effects even in dilute form, (e.g. parathion). The use of ULV (Ultra Low Volume) equipment also results in the direct application of concentrated pesticide. Thus, there is the potential for direct toxic effects in the field.

In most pest control operations on the farm, the applicator is exposed to the second highest amount of pesticide, i.e. second to the mixer/loader. The actual amount of pesticide that the applicator may receive is dependent on the application equipment used, the weather conditions and equipment failure.

Commonly used application equipment can be ranked in terms of highest to lowest exposure of the applicator to pesticide as follows:

fumigant applications,
back-pack sprayers and hand applicators,
airblast sprayers, and
hydraulic boom sprayers (high and low pressure).

The applicator of fumigants within structures (greenhouses, storage areas) probably faces the greatest potential risk. No application equipment, as such, is used with these pesticides as they are usually only available to farmers in aerosols or smoke applicators. If an accident occurs during the set up of these fumigants and the applicator is not able to leave the treated building, significant toxic effects, possibly death, could result. Back-pack sprayers and hand applicators are heavy items of equipment when loaded, and may be difficult to hoist onto the back. During loading, hoisting onto the back and application, these sprayers often leak, resulting in pesticide coming into direct contact with the applicator. In addition, these pieces of equipment are often used inside structures (greenhouses, barns) where sprays may deflect onto the applicator, and the atmosphere contains pesticide droplets or fumes. Complete personal protection equipment should be used in these circumstances or exposure can be severe. Airblast sprayers are usually used in the application of pesticides in orchards or vineyards. These sprayers use high velocity air to carry pesticide into the foliage. Often the applicator is covered with spray mixture as it deflects off the foliage, or is blown back by wind. Complete personal protection equipment should be worn. The hydraulic boom sprayers are the most commonly used sprayers on most farm operations. While the applicator does not usually receive as great an exposure as with the other spray equipment, exposure still occurs.

Weather conditions for application with airblast or hydraulic boom sprayers that would minimize applicator exposure are low winds blowing across the field perpendicular to the direction of travel by the sprayer. If the farmer commences application at the downwind edge of the field and proceeds upwind, there will be minimal exposure due to passage through the spray cloud. This situation is relatively rare and farmers often have to drive through the spray cloud repeatedly during the application. During application, low temperatures are preferable to high temperatures as volatilization of the pesticide will be at a minimum and farmers are more likely to wear personal protection equipment.

The applicator may be directly exposed to the spray mixture in the field if there is equipment failure. High pressure lines containing the spray mixture may rupture or their couplings fail. As these lines and couplings are often located directly behind the applicator, this may result in the applicator being soaked by the spray mixture. A regular maintenance program for sprayers and the use of lower pressures would reduce the chance of this exposure. The clogging of nozzles of hydraulic boom sprayers is a common problem, especially if wettable powder formulations are in use. The farmer may remove the nozzle and attempt to clean it by using a readily obtained source of air pressure, the lungs. This requires the nozzle to be placed against the lips, with the potential of ingestion of the spray mixture. This source of exposure could be reduced if the use of wettable powder formulations was minimized, and if nozzle cleaning equipment was kept with the sprayer in the field.

During the application operation, the applicator may wish to eat, drink, smoke or attend to bodily functions. These actions may result in the ingestion of pesticide and/or the intimate contact of pesticide with the skin. Farmers usually wash their hands before most of these activities, however, smoking is often done without any attempt to clean the hands. If the "coffee break" is taken in the field, clean water is usually not available for washing. If food is stored on the tractor or in the area of the treated field, it may be contaminated with pesticide.

The aerial application of pesticides is usually performed by a contracted applicator with the timing of the applications and pesticide selection decided by the farmer. The hazards to the aerial applicator are very similar to those experienced by the ground applicator with the additional hazards associated with aircraft used at low altitudes, (e.g. power and telephone lines and other obstructions).

The potential exposure situations to farm personnel are generally the same as with ground application of pesticides with the following exceptions:

Transport of the pesticide may be by the farmer or the pilot and the pilot's assistants.

The farmer is usually not involved in the mixing/loading, rather the pilot or the assisting personnel do this operation.

Drift may be a more significant problem from aerial applications.

The pesticide is usually in a relatively concentrated mixture in the aircraft's spray tanks since ULV equipment may be used.

Mechanical failure of the aircraft may result in the emergency dumping of fairly concentrated pesticide into non-target areas.

Flagmen, although now rarely used, may be directly exposed to the spray mixture.

Statistics on the exposure of farm workers to pesticides during mixing and application are not well documented in Ontario (or in Canada for that matter) and only a small number of scientific studies have been conducted under Canadian conditions. Most of these studies have not measured contact per se but have concentrated on the detection and

measurement of the pesticides in body fluids, an indirect measure of contact as well as absorption.

Franklin et al (1981) studied exposure of orchard workers to azinphos-methyl under actual use conditions in the Okanagan Valley during the use of ultra-low volume airblast spraying equipment. In all cases, metabolites of the insecticide were found in urine and a good correlation between total amount sprayed and the amounts of metabolite excreted in the urine over the 48 h post-spray period was observed. Measurement of skin contact by the use of absorbant patches showed that the major sites of contact were the normally unprotected skin of the hand, face and neck region. Air concentrations of the insecticide would have resulted in a mean uptake of 0.024 mg/h had the workers not worn respirators. Dermal contact resulted in the presence of 100 to 400 µg of azinphos-methyl metabolites in urine. In all cases exposure did not result in symptoms of poisoning or in lowering of serum or plasma cholinesterase activity. The type of protective clothing worn did not seem to have a significant effect on the degree of exposure in this type of application. A similar study of herbicide aerial applicators in Saskatchewan (Franklin et al, 1982) using urinary excretion as an indicator showed that highest exposure occurred in association with mixing of the herbicide. The pilot of the spray-plane showed the next highest exposure while that in the water carriers and the flagmen was lowest. Amounts of 2, 4-D metabolites excreted were equivalent to 39, 9.7, 4.25 and ca. 4.0 µg/kg (body weight) respectively.

In studies on herbicide applicators working on transmission line rights-of-way in Ontario, Libich et al (1984) also showed by using urine levels of herbicide metabolites that skin was a major route of absorption of the herbicides. Use of a hand gun resulted in less total exposure than a mist blower and also reduced the amount of herbicide entering the body through the inhalation route. Frank et al (1984) studied exposure to 2,4-D by measuring urinary metabolites in forest workers involved in

aerial spraying. They found that exposure could be reduced by wearing a full set of protective clothing. 2,4-D and its metabolites were also found in urine during the pre-spray period and swabs of equipment and living quarters showed contamination of areas such as vehicles, refrigerator door handles, desks and underlines the importance of adequate cleanup after spraying or handling equipment.

Maddy (1981) reported a total of 116 occupational injuries in persons involved in mixing and loading pesticides in California. Slightly more than half the cases involved aerial applications of pesticides and, of the total, 42% caused systemic illness and 27% each caused eye and skin injuries. Mevinphos and methomyl accounted for 40% of the systemic injury, sulphur was the cause of 25% of the eye injuries and Omite caused half of the skin injuries. In some cases, poisoning was due to accidental exposure to the pesticide during leakage or rupture of equipment but in many cases no specific contamination incident took place.

In a study of Swedish farmers and professional spray applicators using phenoxy herbicides such as 2,4-D, Kolmodin-Hedman et al (1983) showed contamination by measuring concentrations in air and in urine. In all cases they were able to show exposure but it was seen to be more severe in the case of sprayers who took few precautions than in those who were careful. High exposure also resulted from mixing of powdered formulations of the pesticides and the major route of absorption appeared to be the skin, with inhalation and swallowing of the spray droplets as secondary routes of absorption. Stephen and Davis (1981) observed similar high contact levels for captan powder used to treat seed potatoes during cutting in North America. Again, respiratory exposure, as measured with a respirator, was considered high, although no information on total amount in the body was given.

Studies of paraquat and diquat applicators in Florida (Wojeck et al, 1983) again showed that dermal contact with the pesticides was the major source of exposure. Air contamination represented less than 1% of total exposure as measured by respirator cartridge and pads attached to the outside of the clothing. Paraquat was only detected in the urine of one worker on one day suggesting that penetration through clothing and skin was minimal.

Exposure After Application

Contrary to label directions, farmers and farm labourers may re-enter treated areas before minimum re-entry periods have expired. Exposure risk is highest in fruit and vegetable production where hand labour is used to a major extent in weeding and harvest operations. The greatest risk to farm workers is caused by those pesticides with high toxicity and long residual lives in the field, especially those that are applied as wettable powders.

This problem has been recognized for some time and has been the subject of recent extensive reviews (Gunther et al, 1977: Popendorf and Leffingwell, 1982). Toxicological problems concerning re-entry have almost always been associated with organophosphorus pesticides (Kahn, 1980). Although traditionally associated with pesticides applied to foliage in hot and dry areas such as California (Gunther et al, 1977), it may also be found in areas with similar climate to Ontario (Bogden et al, 1975) and soil residues of the pesticides may also be important as a source of toxicant (Spencer et al, 1975 and 1977).

Popendorf and Spear (1974) showed that pesticide contaminated dust dislodged from leaves in California grape and peach orchards was a major source of contamination with pesticides, either through contamination of skin or inhalation and subsequent ingestion of the dust particles.

Re-entry poisoning incidents in Canada and Ontario are not well documented and no reports of poisoning could be found. McEwen et al (1980) have studied the dissipation of pesticides from sprayed crops under Ontario conditions and showed significant differences between crops. Parathion dissipated rapidly from onion and carrot leaves with less than 25% remaining 8 hours after application while the same pesticide took about two days to dissipate to 25% of initial concentration in apple leaves. The amount of residue which could be dislodged followed similar trends. The authors point out that the persistence of pesticide residues on foliage is very dependent on climate and other factors and suggest that more information should be sought under Canadian conditions.

Bystander Exposure

It is impossible to apply a pesticide from ground or aerial sprayers without some drift. Drift from aerial applications may be more severe than that resulting from ground applications. As the farmhouse is often immediately adjacent to the treated areas, the people in its environs may be exposed to this drift. Where treated areas are adjacent to urban environments, the drift may result in pesticide exposure of people in the urban setting. No data on this type of exposure in Ontario is available.

Factors which reduce drift include: application at wind speeds no greater than 11 km/h; use of low pressures which increases droplet size and minimizes the time the droplets stay in the air; application during lower air temperatures; the use of low volatility formulations; and minimizing the distance between the spray nozzles and the target. These procedures reduce both applicator and bystander exposure.

Disposal of Containers

The final operation which would expose farm personnel to pesticides is the disposal of empty pesticide containers. These containers may be disposed of on the farm by burying or burning, or in the local sanitary landfill site. In the case of liquid pesticides, farmers may not rinse the containers thoroughly during the mixing, resulting in a significant pesticide residue remaining in used containers. Often, in the case of dry materials, a residue of dust is left in the carton or bag. During the course of disposal, this pesticide may be released and cause human exposure. This is especially true if cartons or bags are incinerated and farm workers exposed to the smoke and combustion byproducts. In addition, the recommended practice for the disposal of metal containers suggests puncturing the container. This may result in direct exposure of the farmer to any residual pesticide in the container. Farmers should thoroughly rinse all containers prior to disposal and wear personal protection equipment during disposal. Use of special rinse equipment is recommended.

Failure to dispose of pesticide containers properly has resulted in their use in dangerous situations. For example, empty pesticide containers have been used to store foodstuffs and pesticide drums have been used as floats on rafts and docks. The disposal of pesticide containers by municipal garbage removal has resulted in the exposure of sanitation personnel to pesticides either when loading the disposal trucks or when the containers are buried.

Discussion

Many of the safety procedures mentioned herein are already the subject of pesticide regulations which are difficult, if not impossible, to enforce at the farm level. Those farmers who have had personal experiences

involving pesticide poisonings are much more likely to adhere to proper safety practices.

The evidence in the literature indicates that greatest exposure of farm personnel to pesticides occurs during the mixing/loading operation. Thus methods to reduce exposure via this pathway would be a priority of improved safety practices. It is noted that inadequate attention is given to:

Compliance with existing Ontario regulations with regard to the transportation, storage, mixing, application and disposal of pesticides.

The availability, knowledge and awareness of all practical measures to avoid contact with pesticides.

The use of currently available and recommended personal protection equipment.

Increasing awareness of closed-loading systems, particularly for use with formulations of pesticides which form dusts.

The use and availability of adequate methods for clean-up of pesticide spills at the mixing/loading site and the use and availability of facilities for maintenance of proper cleanliness of person and clothing.

The availability of soluble granule rather than wettable powder formulations of pesticides.

Routes of Entry of Pesticides Into the Body

Pesticides may be absorbed into the body as a result of contact with the skin (dermal absorption), inhalation and/or ingestion.

Dermal

Following the penetration of toxic chemical through the skin and subsequent distribution through the body, systemic poisoning may occur. The resulting effects depend on the toxic action per se of the substance and on the amount and rate at which it is absorbed from the site of contact. The spraying or dusting of pesticides has been shown in some cases to result in exposure of skin to 20-1700 times the amount reaching the respiratory tract. The quantities vary with the working concentration, techniques of application, duration of exposure and the protective clothing used.

Polar and non-polar pesticides diffuse through the stratum corneum of the skin by different molecular mechanisms. Polar molecules pass through the watery parts of the intercellular area. Organophosphorus compounds, organochlorines, nitrophenols, nitrocresols and anilines have high skin permeability.

Lipid soluble pesticides are more readily absorbed. Emulsions are more easily absorbed than are wettable powders. Pesticide formulations with pH less than 5 and higher than 8 are irritants: the alkalis are keraten solvents and the acids are protein precipitants and both destroy the stratum corneum of the epidermis. Volatile active ingredients persist as vapours and are readily absorbed through the skin.

Formulations containing petroleum products, emulsifiers, surfactants or organic solvents such as xylene, alcohols and ketones are much more hazardous than the same products in granulated form (Bainova, 1981). Some pesticides may persist for long periods in the skin and accumulation may lead to dermal toxicity and the development of delayed skin irritation related to repeated applications. Chlordane has been reported to be able to persist on skin for at least 2 years, methoxychlor, captan and malathion, 7 days, and endosulfan, dicofol,

parathion and phosmet from 1 to 112 days (Kazen and Bloomer, 1974).

Pesticides deposited on the skin may diffuse through the epidermal barrier into the capillaries of the skin and reach the circulatory system, producing systemic toxicity. They may also produce various forms of dermatitis.

Environmental factors and social habits play a large part in predisposition to pesticide toxicity. Dermal absorption is increased with high temperatures and humidity and other factors which lead to perspiration. This is particularly important in workers in greenhouses. Those who have poor cleansing habits, disregard protective clothing, or wear soiled clothes for prolonged periods of time are frequently the victims of dermal pesticide absorption and poisoning.

Inhalation

Pesticides may be inhaled as gases, vapours, small droplets of liquid, smoke composed of solid particles or as dust. In some circumstances, the inhalation risk may not be recognized until poisoning has already occurred. This most frequently occurs with pesticides which are volatile or formulations such as granules which include a portion of their contents as very fine particles. Examples of poisoning not recognized at the time have occurred among workers filling hoppers or attending machines for seed dressing with toxic materials, or handling fumigated grains which have not been adequately ventilated before handling (Kaloyanova, 1981).

Pimentel and Marques (1969) and Pimentel and Peixoto Menezes (1975) reported cases of lung disease associated with the use of copper compounds (Bordeaux mixture) for the control of the fungal pathogens in vineyards in Portugal. These lung lesions were found to contain copper and were also associated with liver disease in which copper containing

lesions were also present (Pimentel and Peixoto Menezes, 1977). Lings (1982) reported an increased incidence of reduced lung function, fibrosis and changes in the lung tissue of farmers in Denmark who had used "biocides". The pesticides involved were not identified except for the statement that 156 different compounds were used by the farmers.

Ingestion

Ingestion of pesticides is not considered a very common cause of occupational poisoning by pesticides, and is usually the result of the use of soft drink bottles and kitchen utensils for the storage of liquid concentrates. Poisoning has also occurred by using the mouth to blow blocked nozzles free of blockage and from consuming food grossly contaminated with pesticide residues.

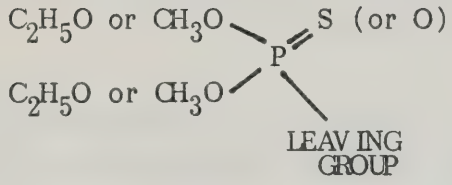
Toxicology of Major Pesticides Used in Ontario

This section of the report summarizes available published information on the toxicology of pesticides used in Ontario. For the most part, this information is obtained from studies in laboratory animals (though some human studies are available) and is required by Government as part of the pesticide registration process. The data presented in this section, along with confidential proprietary information, are used by regulatory authorities to determine human health risks associated with intended use of pesticides, the development of appropriate label warnings and other necessary control procedures.

The pesticides used in Ontario may be divided into a number of major classes by virtue of their mechanism of action and their chemical structure. In the following section these pesticides are discussed by major class. Those pesticides listed in Table 1-2 are discussed in more detail with respect to the specific hazard involved in their use.

Organophosphorus, Cholinesterase-inhibiting Pesticides

General Chemical Structure



Common Commercial Organophosphorus Insecticides

These pesticides may be ranked with respect to their toxicity to test animals and are listed below in approximate order of descending toxicity. "Highly toxic" organophosphorus compounds have listed oral LD_{50} values of less than 50 mg/kg (body weight). Some of these chemicals are systemic: i.e., they are taken up by the plant and translocated into foliage and sometimes into the fruit. These compounds are marked by an "*".

Highly toxic: phorate, disulfoton *, fensulfothion, demeton *, terbufos, methidathion, chlorfenvinphos, parathion, azinphos-methyl, methamidophos, isofenphos, famphur.

Moderately toxic: dichlorvos, demeton-methyl*, chlorpyrifos, phosalone, dimethoate, diazinon, naled, trichlorfon, acephate, malathion.

Organophosphorus compounds are used mainly as insecticides and their usage has increased since the ban on certain chlorinated hydrocarbons. In 1983 about 340,000 kg of organophosphorus compounds were used in agriculture in Ontario. The major advantage of these chemicals is that they generally do not accumulate in the environment as some of the

cholorinated hydrocarbons have. Their major disadvantage is that they are far more acutely toxic, and are probably the most common cause of occupational pesticide illness. The world literature is replete with records of massive poisoning by these pesticides (Hayes, 1978: Knaak et al, 1978: Namba et al, 1971) although the situation in Ontario seems to be less serious (see Tables 3-2 and 3-3).

The major route of exposure to this class occurs through the skin. The principal mechanism of action in organophosphorus poisoning is inhibition of cholinesterase, the enzyme performing the hydrolysis of acetylcholine to choline and acetic acid. Acetylcholine acts as a mediator of nerve impulses from nerve fibers to other nerves or organs. The process of transfer of the nerve impulse is very short, lasting about 0.002 of a second, because the acetylcholine is rapidly removed from the site through the action of cholinesterase. Organophosphorus compounds react chemically with the enzyme causing inactivation which, in turn, causes accumulation of excessive quantities of acetylcholine in parts of the nervous system and leads to elevated concentrations in plasma and interstitial fluids. Organophosphorus poisoning results in the following reactions:

1) Effects due to overstimulation of nerves to lungs, gastrointestinal system, heart, kidney, sweat glands, pupil and muscles.

2) Central nervous sytem effects due to excess acetylcholine acting on receptors of the nerve cells in the brain and effects on other enzymes which may also be inhibited by organophosphorus compounds.

Poisonings by all organophosphorus compounds are basically similar, but the severity of the condition is, to a degree, dependent on the rate at which the specific organophosphorus compounds are metabolized in the body, especially by hydrolysis in the liver, and thus limiting the amount

of pesticide available to inhibit cholinesterase enzyme in other tissues. Acute poisoning is the most frequent type of intoxication since organophosphorus compounds do not accumulate readily. Symptoms are usually seen from four to twelve hours after exposure and all organophosphorus compounds reveal similar signs although of different intensities.

The nervous system effects of cholinesterase inhibition are usually related to stimulation of increased bronchial salivary, lacrimal and sweat glands, resulting in increase of lacrimation and sweating and in tightness in the chest, with prolonged wheezing, breathing difficulties, slight pain in the chest, increased bronchial secretions and cough followed in severe cases by edema of the lungs. Other effects are seen in muscular weakness, convulsions, coma, loss of reflexes and, in some cases, irregular heartbeat and cardiac arrest.

Chronic poisoning has rarely been reported, however, Davignon, et al (1965) indicated that, among a group of 140 workers who used organophosphorus compounds in their occupation, there was a greater incidence of miosis (contraction of the pupil), decreased reflexes, tremors and disturbances of equilibrium than a control group having no contact with insecticides. They also noted a time-related connection with the duration of exposure in affected workers.

The development of delayed neurotoxicity in association with organophosphorus compounds is distinctly different from the acute toxicity seen as a result of cholinesterase inhibition. The syndrome is usually not seen for up to 14 days after exposure or after the patient appears to have recovered from an acute poisoning episode. At the onset of the symptoms, weakness and a lack of muscular control predominate, usually developing first in the lower limbs and then progressing through paralysis which may even affect the upper limbs. Not all organophosphorus compounds appear to be capable of producing

delayed neurotoxicity. No organophosphorus compounds which are known to cause delayed neurotoxicity in man or animals are currently used in Ontario. Examples of pesticides from this class are discussed below.

Parathion

Parathion is a broad spectrum insecticide used in the production of various fruits and vegetables and corn. It is a contact and stomach poison and is sold in Ontario as a wettable powder, emulsifiable concentrate or flowable liquid, from which dilute sprays are used for application. Parathion has been the cause of hundreds of human poisonings on a worldwide basis, many of which were fatal. These have occurred from occupational contact (Grob et al, 1950: Melby et al 1964), suicidal use, and accidental ingestion.

Environmental Factors Affecting Toxicity

In the environment, parathion undergoes a number of important chemical conversions. The first is conversion to paraoxon, which is about three times more toxic, and five times more rapidly absorbed, than the parent compound (Nabb, et al, 1966). Paraoxon is later hydrolyzed to diethyl phosphoric acid or is degraded to aminoparathion which is devoid of biological activity (Menzie, 1969). Studies on environmental contamination with parathion in various areas of Ontario have been carried out. In 1975-77 no parathion was detected in rain water, stream water, or in stream bed sediments in 11 agricultural watersheds in Southern Ontario where parathion was being used (Braun and Frank, 1980). However, parathion was detected in surface soils of 31 apple orchards and 16 sweet cherry orchards at maximum levels of 0.021 and 0.006 mg/kg. (Frank et al, 1976). Residues of parathion have also been found in soil after repeated application for onion maggot control (Sethunathan et al, 1977). These levels, however, are hardly likely to present an occupational hazard.

Absorption, Distribution, Excretion and Metabolism

Parathion is absorbed through the skin, the gut and by inhalation (Frederickson, 1961). In occupational poisoning the most common route is through the skin. The average rate of dermal absorption has been considered to be 0.059 mg/min/cm^2. In the case of oral ingestion parathion is very readily absorbed through the gut and many poisonings have been caused by this route. The vapour pressure of parathion is so low that exposure to vapour alone is not likely to cause poisoning, but inhalation of fine dusts or wet particles may be extremely hazardous. Once absorbed, it has been shown in rats that the compound accumulates to some extent in the cervical brown fat and salivary glands, to a marked degree in the liver, kidney, fat tissues, and to a fairly high degree in the gastric and intestinal walls, thymus, adrenals, skin, bile and intestinal lumen. Only very small amounts are found in nervous tissue, muscle and bone marrow (Frederickson and Bigelow, 1961). Parathion is metabolized primarily in the liver but also in the brain and lungs (Norman and Neal, 1976), and is converted into the more toxic paraoxon, and other less toxic compounds. Further metabolism occurs and the major metabolites recoverable in the urine are paranitrophenol and diethylphosphate, 86% of the former being excreted in 8 hours while the excretion of the latter is much more protracted.

In human studies involving the application of 5 g of 2% parathion dust to the hands and forearms of volunteers at different ambient temperatures, it was found that the maximal excretion of the metabolite paranitrophenol occurred within 5 to 6 hours. Furthermore, the absorption, and consequently the excretion, was proportional to the temperature. The average recovery was 0.196 mg/person at 14.4^{o}C, 0.246 mg/person at 21.1^{o}C and 0.287 mg/person at 27.8^{o}C and 0.804 mg/person at 40.6^{o}C.

Toxicity in Animals

The LD_{50}s of parathion of unspecified purity were summarized by the National Institute for Occupational Safety and Health in 1976: the oral LD_{50} was 13 mg/kg (body weight) in male rats and 3.6 mg/kg (body weight) in females. The respective dermal LD_{50}s were 21 and 68 mg/kg (body weight). The intraperitoneal LD_{50} was 7 mg/kg (body weight) in male rats and 4 mg/kg (body weight) in females, while in mice it was 9-10 mg/kg (body weight). The single dermal LD_{50} in rabbits was 40-50 mg/kg (body weight). The intravenous LD_{50} was 3-5 mg/kg (body weight) in cats and 12.20 mg/kg (body weight) in dogs. The acute toxic effects observed involved sweating, salivation, diarrhea, bronchoconstriction, muscle fasciculation and coma. The cause of death appeared to be primary respiratory failure (Taylor, 1980).

The maximum concentration which had no effect on red cell cholinesterase activity in pigs and rats was found to be 1 mg/kg (body weight) and 0.02 mg/kg (body weight) respectively over a period of 25-122 days (Edson, 1964).

There are some reports of chronic effects of parathion and other organophosphorus insecticides, for example, parathion, diazinon and trichlorfon have been reported to have teratogenic potential, (Kaloyanova, 1981), however, these findings have not been repeated in the open literature. The International Agency for Research on Cancer has assessed the results of the four long-term studies available on parathion in rats and mice and found that "although a dose-related increase in the incidence of adrenal cortical adenomas was observed in male and female rats of one strain, the significance of these lesions in aged rats is not well understood. The low incidence of carcinomas at this site was observed in each of the treated groups in animals of both sexes. The other experiments in rats and mice were considered to be inadequate for evaluation".

Effects on Reproduction

It has been shown that radioactive parathion passes through the placental barrier in pregnant rats and that toxic doses may cause prenatal and postnatal death of the young and reduce their weight gains. The fetus appears to be more sensitive than pregnant adults since the inhibition of cholinesterase activity in fetal blood was lower than in the parents. Infants are more susceptible probably due to their poorly developed microsomal system. In these and other studies, no teratogenic effects have been found.

Mutagenicity Testing

No evidence of mutagenicity was found in a series of tests including micro-organisms, cultured mammalian cells, Drosophila melanogaster or in dominant lethal tests in mice (Hayes, 1982).

Toxicity in Man

In the USA, parathion has been the main cause of cropworker poisoning (Quimby and Lemmon, 1958). Workers may be affected after working in a field for only a few hours when days or even weeks have elapsed since the insecticide was applied. On occasions, whole work crews have been affected, some fatally. However, some states such as California have, by means of controls on re-entry times for workers and other regulations concerning parathion, reduced the incidence of poisoning requiring hospitalization from 275 cases in 1959 to 55 in 1970. Most cases have been due to dermal absorption. Manifestations of acute poisoning in humans by parathion are similar to those in animals.

In dose titration studies in human volunteers, it was found that consumption of 0.05 mg/kg (body weight) parathion produced no depression of red cell or plasma cholinesterase and no symptoms of illness, while repeated daily doses of 6mg/kg (body weight) slightly

inhibited plasma cholinesterase (Rider et al, 1969).

Phorate

Phorate is a systemic and contact insecticide and acaricide. This organophosphorus compound is used to protect potatoes, lettuce, corn and rutabagas from insects, mites and certain nematodes. It is sold in Ontario in the form of 15% granules. This substance is extremely toxic but there appear to be few reported cases of poisoning in man due to its use.

Environmental Factors Affecting Toxicity

Phorate is unstable to hydrolysis and the half-life at pH 8.0 and 70°C is only about two hours. However, the compound is metabolized by plants to very potent anticholinesterase agents including the sulfoxide and sulfone of both phorate and its oxygen analogue. Because of this, plants and soil to which phorate has been applied increase in anticholrinesterase activity for several days and then slowly decline over several weeks.

Absorption, Distribution, Excretion and Metabolism

Phorate is rapidly absorbed by the oral and dermal routes. Studies in rats and cows indicate that they metabolize phorate in a way similar to plants. In animals 0,0-diethylphosphorothiolic and later 0,0-diethylphosphoric acid are formed and excreted in the urine. Phorate appears to have an affinity for liver and kidney and, in studies using radioactive material, most of the radioactive material was found bound to these tissues.

Toxicity in Animals

As with other organophosphorus compounds, the effects of poisoning with this substance involves anticholinesterase activity. It is very toxic, the oral LD_{50} in rats being about 1mg/kg (body weight) in females and 2 mg/kg (body weight) in males. The dermal LD_{50} in female rats is about 2.5 mg/kg (body weight) and in males about 6.2 mg/kg (body weight) (Gaines, 1960). The dermal absorption is very rapid and treated animals may die within an hour or two following application. The most toxic metabolite, the sulfone, is about twice as toxic as the parent compound.

Published data on subacute and chronic toxicity does not appear to be available, but phorate was not mutagenic in bacterial systems (Simmon et al, 1976) and did not produce dominant lethal mutations in mice (Jorgenson et al, 1976).

Toxicity in Man

Those few cases of poisoning in man which have been reported involved those who planted treated seeds, cleaned planting machines or carried out related tasks while neglecting recommended protective measures. An anecdotal case is reported of a woman thought to have significant symptoms of poisoning ten hours after she had travelled for two hours in a truck cab containing a sample of phorate contaminated soil in a polyethylene bread wrapper. Recovery occurred following atropine treatment.

Demeton

Demeton is an organophosphorus systemic and contact insecticide used in fruit and vegetable production. It is sold in Ontario as an emulsifiable concentrate. It is highly toxic both by oral and dermal routes and a number of cases of dermal exposure have been reported. The mechanism of action is the inhibition of acetylcholinesterase.

Absorption, Distribution, Metabolism and Excretion

Demeton is well absorbed by all three routes. Metabolism converts this chemical first to the sulfoxide then the sulfone, both of which are as toxic as demeton.

Toxicity in Animals

The oral LD_{50} in male and female rats is 2.5 and 6.2 mg/kg (body weight) respectively. The corresponding dermal values are 14 and 8.2 mg/kg (body weight) (Gaines, 1960). Levels as low as 1 ppm (1 mg/kg body weight) have been shown to reduce blood and brain cholinesterase by about 10%. The corresponding levels in rats fed 20 ppm were reduced by about 85% and at 50ppm severe poisoning resulted with reductions of cholinesterase levels by over 90%.

Effects on Reproduction

Demeton has been shown to be teratogenic in duck and pigeon eggs but in mice it appears to only be embryotoxic, possibly due to the toxicity in the mother.

Toxicity to Man

In human volunteers 0.05 mg/person/day had no effect on cholinesterase. No illness was produced with up to 6.4 mg/person/day but plasma cholinesterase was reduced by 80% although the red blood cell levels were not significantly reduced (Rider and Moeller, 1964). A number of serious poisoning and deaths caused by demeton have been associated with occupational exposure (Kaiser, 1953: Kleinman, 1960) and the hazard of this chemical seems to be similar to that of parathion.

Azinphos-methyl

This insecticide is widely used in fruit production in Ontario and is available as a wettable powder and emulsifiable concentrate.

Environmental Factors Affecting Toxicity

Azinphos-methyl is a moderately persistent pesticide which is rapidly hydrolyzed by alkali.

Absorption, Distribution, Excretion and Metabolism

Azinphos-methyl is easily absorbed through skin, lung and stomach. Franklin et al, (1981) have studied its uptake and excretion in orchard workers in B.C. (see above). Of the dose applied in man 69% was excreted within 120 hours (Hayes, 1982).

Toxicity in Animals

Acute oral LD_{50} in the rat ranged from 16.4 to 11 mg/kg (body weight) with a dermal LD_{50} of 220 mg/kg (body weight). Oral LD_{50} in the pig was 80mg/kg (body weight) (Hayes, 1982). A two-year feeding study in rats at 5 and 10 mg/kg/day showed little in the way of effects except for depressed blood cholinesterase. No tumors were observed (Hayes, 1982). Dogs fed 1 mg/kg/day showed clinical signs of poisoning. Similar effects were not seen at 0.25 mg/kg/day (Hayes, 1982).

Toxicity in Man

An occupational intake level of 0.03 mg/kg/day has been suggested in man, (Hayes, 1982) however, a number of human poisoning incidents have been reported in the case of accidental contamination.

Disulfoton and Terbufos

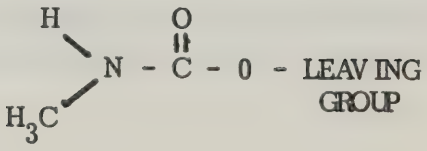
Disulfoton has a high oral toxicity in the rat of 2.5 to 12.5 mg/kg (body weight) (Wagner, 1983) and is rapidly absorbed by all three major routes. Terbufos is also a highly toxic material and, as a result, is available only as a granular formulation for incorporation into soil for corn rootworm control. Both materials present an obvious acute hazard but no information was available to the members of the study team to suggest that long-term effects have resulted from exposure to these materials.

Hazard Evaluation

Of the compounds listed in Table 1.2, azinphos-methyl, demeton, disulfoton, parathion, phorate and terbufos belong to this class. The major hazard associated with these compounds lies in their high acute toxicity and the possibility of poisoning after a relatively small exposure. That these compounds are not a major cause of death, suggests that the regulations which govern their use are satisfactory and that a general awareness of their toxicity exists.

Carbamate Cholinesterase-Inhibiting Pesticides

General Chemical Structure



Common Commercial Pesticide Products

These pesticides may be arranged with respect to their toxicity to test animals and are listed below in approximate order of descending toxicity. "Highly toxic" carbamates have listed oral LD_{50} values less than 50 mg/kg (body weight). Some of these chemicals are systemic: i.e., they are taken up by the plant and translocated into foliage and sometimes into the fruit. These compounds are marked by an "*".

Highly Toxic: aldicarb *, oxamyl *, carbofuran *, methomyl, formetanate HCl.

Moderately Toxic: propoxur, pirimicarb, carbaryl, (Adapted from Morgan, 1982).

Carbamates are used mainly as insecticides. In 1983, about 88000 kg of carbamates were used in Ontario. They may be absorbed by ingestion, dermal absorption and inhalation. The toxicity of compounds in this group varies from high to very low. Dermal absorption is very variable and the high vapour pressure of some of these compounds makes them particularly subject to the effect of temperature when they are sprayed on surfaces (Hayes, 1975). However, the carbamates are relatively safe for man in spite of being effective insecticides (Hayes, 1982).

The mechanism of action of carbamate poisoning is, as in the case of organophosphorus compounds, inhibition of cholinesterase. However, in this case the duration of inhibition is usually briefer, and the effects somewhat mitigated. Since the toxic effects are produced by the accumulation of acetylcholine in the nervous system the symptoms are similar to those described for organophosphorus compounds. Another difference is the relatively wide separation of the smallest dosage which produces mild illness and the dosage of the same compound necessary to produce death. Both of these features have the same pharmacological basis, namely, the relatively rapid spontaneous reactivation of

cholinesterase inhibited by carbamates. This mitigates the effects, but limits the usefulness of blood enzyme measurements in diagnosis of poisoning. A further difference is that, in carbamate poisoning, oximes which are used to reactivate cholinesterase are frequently useless. Carbamates are actively metabolized by the liver and degradation products are excreted by the liver and kidney. The toxicology of several carbamates is detailed below:

Carbofuran

Carbofuran is a carbamate systemic and contact insecticide used in Ontario to protect potatoes, carrots, rutabaga, alfalfa, corn and tomatoes. It is sold as granules and a flowable product. In accidental poisoning it produces mild illness, rapid onset with quick recovery which is characteristic of carbamates.

Environmental Factors Affecting Toxicity

In the environment the chemical has a half-life of 30-60 days varying with the type of soil. On plants no traces of the substance have been found 21 days after application to alfalfa at 0.6 kg/ha.

Absorption, Distribution, Metabolism and Excretion

Carbofuran is well absorbed by the oral and respiratory routes but appears to be poorly absorbed through the skin. The metabolites formed in the body may be excreted free or as conjugates. The chemical is rapidly metabolized by mammals and over 90% excreted in the urine and the half-life is 6-12 hours. When given to cows, about 3% of the dose was excreted in milk, mainly in the first 48 hours after exposure (Hayes, 1982).

Toxicity in Animals

Like other carbamates, carbofuran acts as a reversible cholinesterase inhibitor. It is readily absorbed and shows high toxicity to rats following oral adminstration, however, dermal toxicity is very low. In experimental animals given non-lethal doses, cholinesterase returns to normal in 4 to 6 hours. The oral LD_{50} has been reported by various investigators to be from 8-19mg/kg (body weight) in rats when technical products or 75% wettable powder was used. With 10% granular formulations, this was increased to 132 mg/kg (body weight) (Tobin, 1970). Dermal toxicity, however, is very low, the LD_{50} being $>$ 1000 mg/kg (body weight) in rats and 3400 mg/kg (body weight) in rabbits (Hayes, 1982).

In long term experiments in rats, diets containing 25 ppm had no effect over 2 years. In dogs, diets containing 20 ppm similarly had no effect (Nagnes, 1983). In limited trials using two carbamates, carbaryl and carbofuran, it was reported that they had an effect on the immune system and that both cellular and selected humoral responses were depressed in a dose-dependent fashion (Street and Sharma, 1975). Confirmation of this has not been made by other investigators.

Effects on Reproduction

In a three generation study in rats, a dietary level of 100 ppm (5 mg/kg (body weight)) decreased weight gains of parents and markedly reduced survival of young, but a level of 50 ppm did not injure the adults or young. In dogs diets containing 50 ppm had no effect over one generation. No evidence of teratogenesis was found in rats, rabbits or dogs (McCarthy et al 1971).

Toxicity to Man

A few cases of occupational poisoning in man have been reported resulting in transient blurring of the vision, excessive perspiration and weakness. Recovery was complete in a few hours (Tobin, 1970). Mass but mild poisoning of 142 children has been reported, following the collection of tassels from corn used in the production of seed on the day following accidental spraying of the field with carbofuran. Seventy-four of the children complained of dizziness, nausea and blurred vision. All recovered in 24 hours (Hayes, 1982).

Methomyl

Methomyl is a carbamate insecticide with good contact and oral efficacy. It is used in the production of tobacco, grain, apples, potatoes, tomatoes, rapeseed, lettuce and other field crops. It is sold as an emulsifiable concentrate in Ontario. An earlier dust formulation caused a number of poisonings under field use conditions.

Environmental Factors

Methomyl is readily degraded in the environment with half-life in water of 5 to 6 days (Wagner, 1983).

Toxicity to Animals

The oral LD_{50}s in male and female rats were found to be 17 and 23.5 mg/kg (body weight) respectively. The minimum lethal doses in the guinea pig, dog and monkey were 15, 30 and 40 mg/kg (body weight) respectively. The signs of toxicity included pallor of the eyes, profuse salivation and tremors and death occurred within 15 minutes of administration. Dermal applications of 500 mg/kg (body weight) produced no signs of poisoning in rabbits, however, the same dose was mildly irritating to the skin of the guinea pig. The LC_{50} for rats was

77 ppm in a 4 h exposure to aerosol concentrations of 14 to 144 ppm.

Dogs fed 50, 100 and 500 ppm for 2 years did not develop tumors or show clinical effects, however, males at the 400 ppm level showed a significant increase in testes weight. Two of four male dogs fed on a dietary level of 1000 ppm showed signs of poisoning while two females died. The surviving males showed anaemia, increases in bone marrow cell activity and some bile duct proliferation after two years. The no-effect levels in rats and dogs were 4.9 and 2.1 mg/kg (body weight) respectively (Kaplan and Sherman, 1976).

Absorption, Distribution, Metabolism and Excretion

Methomyl is absorbed by all three major routes. In mammals, the material is degraded into non-toxic breakdown products which are excreted in the urine. Methomyl does not accumulate in the body.

Effects on Reproduction

A three generation study on rats did not show adverse effects at concentrations up to 100 ppm in the diet. Teratogenic effects were not seen in rats fed up to 100 ppm during pregnancy. Mutagenicity does not appear to be caused by methomyl but some concern has been expressed regarding the mutagenic properties of the nitroso derivatives.

Toxicity in Man

Methomyl has been reported as the cause of poisoning in tobacco workers in a case where the application rate was grossly exceeded (Kudo, 1975). Inhalation of a powder formulation has caused some poisonings. Because of high dermal toxicity, methomyl has caused some re-entry poisoning.

Aldicarb

Although not widely used in Ontario (5500 kg in 1983), this material is highly toxic and has caused some concern because it has been found in well water in a number of areas in the USA and Canada, mainly where it has been used on potatoes.

Environmental Factors Affecting Toxicity

Aldicarb is moderately water soluble, is stable under cool acidic conditions and may persist in soil and groundwater for long periods of time. It is used as a soil incorporated, granular formulation in potato and ornamental production in Ontario. Aldicarb has been found in well water in parts of Ontario (Frank, personal communication) and, for this reason, there is some concern although the concentrations reported to date are below thresholds where effects may be expected.

Absorption, Distribution, Metabolism and Excretion

Aldicarb is quite readily absorbed via all three major routes but is also quite easily excreted in the urine, feces or metabolized to carbon dioxide. It is oxidized in the body as well as the environment and the sulphone oxidation product is as toxic as the parent compound. It is also translocated in plants where it has, on occasion, caused poisonings in man through the consumption of accidentally contaminated produce (Hayes, 1982).

Toxicity to Animals

Aldicarb is one of the most toxic carbamate insecticides and has an acute oral LD_{50} of 0.8 to 0.6 mg/kg (body weight) and an acute dermal LD_{50} of 2.5 to 3 mg/kg (body weight) in the rat (Gains, 1969).

Toxicity in Man

Anecdotal reports of aldicarb's high acute toxicity are common in the literature (Hayes, 1982: Aaronson, et al, 1979) but, if treated the effects appear to be short term in nature. No evidence of long term effects have been noted.

Carbaryl

Carbaryl is a carbamate pesticide which is widely used in agriculture and in domestic insect control. It is available as a wettable powder or as a dust and formulations may contain up to 85% active ingredient.

Environmental Factors Affecting Toxicity

Carbaryl is relatively non-persistent in the environment and is rapidly hydrolysed in alkaline environments. It is quite stable in acidic conditions at a pH of less than 6.

Absorption, Distribution and Excretion

Carbaryl is rapidly absorbed via the oral and inhalation routes. Absorbed carbaryl is rapidly excreted, mostly in the urine, or metabolized to carbon dioxide and exhaled. It does not accumulate in the body or in any tissues (Hayes, 1982).

Toxicity in Animals

The oral toxicity of carbaryl is low with reported LD_{50}s in male and female rats of 850 and 500 mg/kg (body weight) respectively (Gaines, 1960). Dermal toxicity is very low with an LD_{50} in rats of more than 4000 mg/kg (body weight) reported by Gaines (1960). In contrast to these, the intraperitoneal LD_{50} in mice is reported as 25 mg/kg (body weight) (Hayes, 1982) which possibly reflects the importance of the liver and gastrointestinal tract as an area of detoxification.

Carbaryl is not regarded as a mutagen (WHO, 1977) or as a carcinogen although it has been reported to stop cell division when used at high doses (Hayes, 1982). Reproductive effects have been noted in some animals and Smalley, et al (1968) reported that it was teratogenic in beagle dogs. That this teratogenic effect was not observed to follow a dose-response suggests that it was an artifact of the assay. Carbaryl also produced teratogenic effects in hamsters but this was at a high dose and may also have been an artifact of the assay. Other animals have not shown similar teratogenicity (Hayes, 1982).

Toxicity in Man

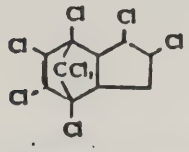
Hayes (1982) has reported few lethal poisonings with carbaryl but some skin reactions have occurred. Sublethal poisonings have occurred mainly due to carelessness. Hayes (1982) reports a case of a man who, in a drunken stupor, consumed 500 ml of an 80% solution of carbaryl. The patient died during treatment, possibly as a result of use of incorrect antidote.

Hazard Evaluation

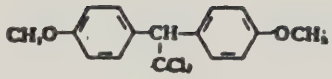
Of the compounds listed in Table 1-2, aldicarb, carbaryl, carbofuran and methomyl are carbamates. Aldicarb is highly toxic by a number of routes of entry and for this reason is available as a granular formulation only. This reduces hazard during handling of the material and the method of use, as a soil applied material, further reduces direct exposure. Although reported to be present in groundwater in shallow aquifers in areas of major use in Ontario (Frank, personal communication), the concentrations are such that no health effects or symptoms are likely to occur. Methomyl and carbofuran have lower but significant acute toxicity. Carbaryl is of low toxicity and is of low hazard under normal conditions of use.

Chlorinated Hydrocarbons

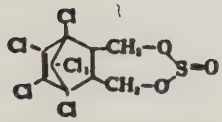
Chemical Structures



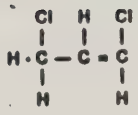
Chlordane



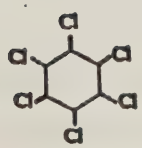
Methoxychlor



Endosulfan



1,3-dichloropropene



Lindane

Common Commercial Pesticide Products

These pesticides may be arranged with respect to their toxicity to test animals and are listed below in approximate order of decreasing acute toxicity.

Moderately toxic: endosulfan, lindane, chlordane, dicofol, methoxychlor, dienochlor and dichloropropenes and propanes (adapted from Morgan, 1982).

Chlorinated hydrocarbons are used in agriculture as insecticides and acaricides to control pests in fruit, vegetable, grain and tobacco products. In Ontario, about 1.4 million kg were used in 1983, however, the vast majority of this was as dichloropropanes and propenes, used as soil fumigants and nematocides. Since some of these chemicals are persistent in the environment after application, many regulatory authorities have sharply curtailed their availability. For example, in the case of DDT, (which is no longer used) persistence in the soil can last for 10 years or more. On the other hand, the soil fumigants are volatile and thus tend to disperse fairly rapidly after application, however, exposure during application could be extensive if precautions are not taken.

Most chlorinated hydrocarbons are efficiently absorbed by the gut, by inhalation and through the skin. The hazard of dermal absorption of these compounds is increased if the pesticide is dissolved in oil or an organic solvent. The lipophilic nature of these compounds enables many of them to penetrate all membranes and become fixed in cells rich in lipids such as the nervous system, liver, kidney and heart muscle. This fixation in tissues varies with the compound used and its rate of metabolism and excretion. Metabolism involves various mechanisms such as oxidation and hydrolysis, and excretion is mainly by the kidney. The toxicity of some of these compounds is given in more detail below.

Endosulfan

Endosulfan is a chlorinated hydrocarbon and powerful contact and stomach poison in insects. It is sold in Ontario as a wettable powder, emulsifiable concentrate and as a dilute dust for the protection of various fruits and vegetables. It is the most acutely toxic chlorinated hydrocarbon insecticide sold in Canada and its persistence in the environment has caused concern in some countries. Its use is limited in Canada pending the production of additional animal toxicity data for regulatory purposes.

Environmental Factors

The main routes of endosulfan dispersion in the environment are volatilization and oxidation to endosulfan sulfate which is as toxic as the parent compound. Endosulfan and its metabolites have been reported to persist in soils for up to two years. The reported half-life of endosulfan in water ranges from one to six months and degradation is by hydrolysis resulting in the formation of alcohol and sulfur dioxide. Braun and Frank (1980) and Frank et al (1980) reported no buildup of endosulfan in soils in Ontario but have reported it as a relatively frequent contaminant of surface water draining from agricultural land (found in 13% of waters sampled).

Absorption, Distribution, Excretion and Metabolism

Absorption is via the oral or dermal route and by inhalation. The uptake in mammalian systems depends on the carrier or solvent used. Alcohols, oils or emulsifiers increase absorption. Generally, it is rapidly absorbed and excreted. The metabolic products are the sulfate, diol, hydroxyether, ketone and unidentified compounds. The rapid excretion appears due to the fact that only about 20% of these metabolites are lipophilic and most are excreted as water soluble substances in the urine and feces after 120 hours (Schupan, et al, 1968). Endosulfan and its metabolites are distributed to almost all organs and skeletal muscles.

Toxicity in Animals

The oral LD_{50}s in male and female rats are 43 and 18 mg/kg (body weight) respectively while dermal values are 130 and 78 mg/kg (body weight). In mice the intraperitoneal toxicity ranged from 6.9 to 7.5 mg/kg (body weight) (Gupta, 1976). The dermal LD_{50} in rabbits is reported as 167 to 187 mg/kg (body weight). Rats administered 5 mg/kg (body weight) per day for 15 days survived but showed liver damage, while increasing the dose to 10mg/kg (body weight)/day produced mortality in some of the treated animals (Gupta, 1976). In cats given 0.5 mg/kg (body weight) repeatedly by intravenous injection, hypertension, papillary dilation, increased cardiac output and an increase in cerebral blood flow were observed. In rabbits, rats and mice, endosulfan toxicity caused hyperexcitability, dyspnea, decreased respiration and tremors followed by convulsions.

Chronic Toxicity

Very little information is available on the chronic toxicity of endosulfan and its metabolites. In 2 year studies on rats fed 10 ppm, the liver and kidney were the organs most affected. The liver lesions consisted of mild degeneration of hepatic cells with eosinophilic cytoplasmic inclusions. The major kidney lesions were dilated tubules, albuminous casts, interstitial nephritis and degeneration of tubule epithelium. Dogs tolerated 30 ppm endosulfan in the diet for 2 years without adverse effects.

Effects on Reproduction

There were no effects on reproduction when female mice were fed 2.5 mg/kg (body weight) per day in three generation studies. However, increasing the dose to 5 mg/kg (body weight) on day 6 to 14 of gestation produced increased mortality of dams and increased rates of absorption and skeletal abnormalities in their fetuses. In a teratogenic study with

pregnant rabbits fed 1.8 mg/kg (body weight), maternal toxicity was evident. However, no significant differences were observed in implantation efficiency, size, sex ratio, and reabsorbed fetuses between control and treated animals. A statistically significant decrease in the weight of testes was found in male rats fed 10 ppm endosulfan in the diet for 104 weeks.

Mutagenicity

Endosulfan has been tested in a number of microbiological screens but results have been inconclusive.

Toxicity in Man

Poisoning in man may be caused by ingestion, dermal absorption or inhalation, the latter two routes being important in occupational disease. Wolfe et al (1972) estimated an endosulfan dermal exposure of 0.6 to 95.3 mg/hour to spraymen applying an 0.08% endosulfan spray. Kazen et al (1974) found that endosulfan persisted on workers' hands has been estimated at 0.01 to 0.05 mg/h using 0.08% spray, however, during mixing operations, 0.18 mg endosulfan was found in a respiratory trap during a five minute mixing operation. Small doses of endosulfan may be fatal. Hayes (1982) reported that one person died after swallowing only "drops" of a formulation. A number of cases of occupational poisoning have been reported in the world literature. Most have occurred while processing formulations. The symptoms usually appeared within two hours of exposure and consisted of headache, restlessness, irritability, vertigo, stupor, epileptiform seizures. In fatal cases there was loss of consciousness, cyanosis, dyspnea and foaming at the mouth.

1,2-Dichloropropane and Related Chemicals

The 1,2-dichloropropanes and 1,3-dichloropropenes are listed in Table 1-2 because of their extensive use, their known carcinogenicity

and their effects on the respiratory system, liver and kidneys. The current manufacture of these chemicals results in a ratio of about 90% 1,3-dichloropropene to 10% 1,2-dichloropropane. The latter material is more environmentally persistent, tends to accumulate in water and may move from the site of application. The 1,3-dichloropropenes tend to break down rapidly in soil if they are correctly applied. There is little information on these chemicals in the literature, however, they are easily absorbed through the lungs, skin and stomach but are readily excreted. In rats 80% of an oral dose was excreted in 1 day and 92% in 4 days. The oral LD_{50}s in male and female rats were reported as 713 and 430 mg/kg (body weight) respectively. The dermal LD_{50} in rabbits was 500 mg/kg (body weight) and it caused severe conjunctivitis if applied to the eye (Hayes, 1982).

Maddy et al (1979) reported that 1,3-dichloropropene concentrations could exceed the allowable concentration of 1 ppm (in air) during agricultural applications. They reported that exposure could be effectively controlled by the use of a suitable respirator and through correct design and maintenance for the application equipment. In man, Torkelson and Oyen (1977) and Hayes (1982) reported that exposure to 1,3-dichloropropene could lead to liver damage, difficulty in breathing and chest pains. Dow Chemical, the manufacturer of 1,3-dichloropropene, has recently informed users that these chemicals cause cancer in laboratory rodents if given by stomach tube (Dow, 1984). Rats were dosed at 25 and 50 mg/kg (body weight) three times per week and mice at 50 and 100 mg/kg (body weight) at similar intervals. At the end of the two year period, the treated animals showed a significantly higher incidence of certain tumors than the control animals. The registration of these and related chemicals is currently under review in the USA and Canada.

Chlorothalonil

This fungicide is widely used in field crops in Ontario. Information on the toxicology of this chemical is not readily available in the open literature and it is difficult to draw conclusions on its toxicology and hazard. The registration of this material is also under active review in the USA and Canada and exposure should be kept to a minimum until further information is available.

Hazard Evaluation

Where these lipid soluble organochlorine compounds accumulate in the various organs, they damage the function of important enzyme systems and disrupt the biochemical activity of the cells. Most poisonings with chlorinated hydrocarbons are chronic in nature and changes in the nervous system are usually the most pronounced effects, producing behavioural changes, sensory and equilibrium disturbances, involuntary muscle activity and depression of vital centres, particularly those controlling respiration. Cardiovascular disturbances and blood diseases have also been reported in the case of some chlorinated hydrocarbons. Many of the effects are reversible at the early stages if exposure ceases.

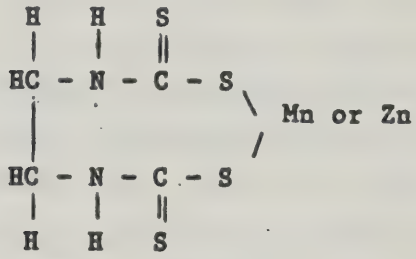
In agricultural practices, acute toxicity is the exception and its rare occurrence is related to misuse or to accidents. Certain chlorinated hydrocarbons appear to have special propensities for specific pathological conditions. For example, chlordane has apparently induced a few cases of self-limited megablastic anemia after protracted low level exposure, however, use in Ontario is currently low and appears to be mainly in the control of structural pests where the infrequent applications reduce the possibility of contact. The reports of carcinogenicity in the dichloropropanes suggest that exposure to this chemical should be avoided unless absolutely necessary and, if used, every safety precaution should be rigidly adhered to until more information is available.

Dithiocarbamates

These compounds are used in agriculture as broad spectrum fungicides. In Ontario about 260000 kg were used in 1983. Dithiocarbamates can be divided into these classes:

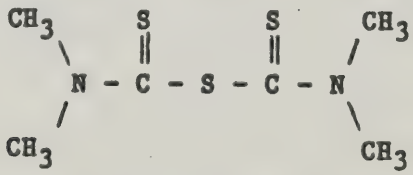
General Chemical Structure and Commercial Products

Ethylene BIS Dithiocarbamates



Commercial products in this class are maneb and zineb. These fungicides are often combined with thiram and with various inorganic salts of copper, manganese, and zinc in commercial preparations. Mancozeb is essentially a combination of maneb and zineb.

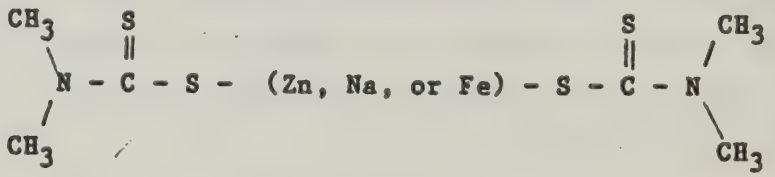
BIS Dithiocarbamates



THIRAM

A commercial product in this class is thiram.

Metallo BIS Dithiocarbamates



Commercial Products in this class are ziram, metiram and nabam, all of which are fungicides.

Dithiocarbamates appear to affect biological systems, mainly as the metabolic products ethylenethioammoniumsulfide, carbon disulfide, ethylene thiourea and isocyanates, which are more biologically effective and more toxic to bacteria, fungi and mammals, than is the primary product. The exact mode of action is not well known nor are the metabolic pathways fully understood, but it appears that carbon disulfide and its metabolites are the only compounds common to the metabolism of all dithiocarbamates and the similarity between poisoning by the various dithiocarbamates and hydrogen sulfide has been noted (Kane, 1970). The property of dithiocarbamates to chelate metals may also play an additional part in their toxicity.

The dithiocarbamates have low acute toxicity potential, however, in the case of some products, heavy occupational exposure has resulted in serious dermatitis, respiratory problems, enteric disease and muscular weakness. The structural relationship of ethylene-bisdithiocarbamate and antabuse has led to typical antabuse reactions on a few occasions, where applicators have indulged in alcohol. Experimental animal studies suggest possible potential for chronic effects from some agents. In addition to these symptoms, zineb and maneb have shown a potential for allergic skin reactions (Bilancia, 1964). Epidemiological studies involving human exposure to thiram have reported ophthalmological lesions with long-term conjunctivitis, reduced visual activity, delayed adaptation, reduced corneal sensitivity and increased pressure in the retinal artery as general symptoms (Ivanova-Tchemeshanska, 1981).

Long-term treatment of laboratory animals with certain dithiocarbamates has caused some concern. In rats high doses of zineb and maneb have caused disturbance in liver and kidney function and paresis (Ivanova-Tchemeshanska, 1981). These pesticides, when used at

half the LD_{50} dose in laboratory animals, have produced inhibitory effects in the thyroid glands. Maneb has also been shown to be teratogenic at extremely high dosages and some degradation products and the ethylenethiourea metabolite common to all ethylene bisdithiocarbamates have proven to be carcinogenic in mice, hamsters, and rats (Ulland, 1972). Maneb and zineb have also been shown to damage the gonads of rats of both sexes (Ivanova-Tchemeshanska, 1981). Of the compounds in Table 1-2, mancozeb, maneb and metiram are probably of major concern because of their high volume of use and the increased possibility of exposure. The toxicology of maneb is detailed below by way of example.

Maneb

Maneb is a dithiocarbamate fungicide used for the production of various vegetables, especially potatoes and tomatoes. It is sold in Ontario as a wettable powder and as a dust. Use experience with this chemical has been good and reports have concerned mainly a few cases of dermatitis and allergic dermatitis.

Environmental Factors

Maneb is unstable in the environment and is easily degraded into a number of other byproducts such as ethylene, ethylenethiourea monosulfate, ethylenebis-diisothiosulfide and ethylenethiourea disulfide. The application of heat will convert some of these substances to ethylene.

Absorption, Distribution, Excretion and Metabolism

Maneb is poorly absorbed from the alimentary tract and following oral administration in mice about 90% was found in the feces and only about 10% in the urine. In rats, the metabolites found in urine and feces

consists of ethylenediamine, ethylene-bis-thiourea and other unidentified metabolites. Ethylenethioureas (ETU) are known to be goitrogenic and suspected carcinogens. Maneb was not found in any of the tissues of rats fed high dietary levels of the chemical for 2 years.

Toxicity in Animals

Maneb possess very low acute oral toxicity and the LD_{50} in mice is 4100 mg/kg (body weight). In rats it is also greater than 4000 mg/kg (body weight). However, maneb intoxication increases thyroid function in rats (Ivanova-Tchemeshanska, 1981). Following a large dose, rats and mice show ataxia and hyperactivity followed by inactivity, loss of muscle tone and alopecea. Most deaths occur within 1 to 2 days.

Twenty-four mg/kg (body weight) fed to rats for 30 days produced a significant increase in thyroid weights, however 20 times this level was required to produce histologically discernible thyroid hyperplasia (Smith et al 1953). The dermal toxicity in these species was too low to measure.

Chronic Toxicity

In a 2 year study, the dietary level of 10000 ppm increased mortality in female rats; the results were equivocal at 5000 ppm but neither level increased the mortality in males. Dietary levels up to 10000 ppm for one year did not affect survival, growth or haematological findings in dogs. In 2 strains of mice, the maximal tolerated dose of maneb was not tumorigenic (Innes et al 1969). A second study in mice revealed a significant increase in lung edema in one strain but not in another (Balin, 1970). As indicated by a number of animal experiments, the characteristic lesions produced by high levels of maneb appear to be enlargement of the thyroid gland, reduced uptake of iodine, and sometimes injury to the testes.

Effects on Reproduction

When maneb was given to rats every other day at a rate of 50 mg/kg (body weight), the incidence of resorption during pregnancy, stillbirths and non-viable young were doubled (Martson, 1969). Fertility was decreased in both male and females when they were fed maneb for one month and then in 1.5 months mated to animals on control feed. When maneb was administered to female rats on day 11 and 13 of organogenesis, congenital abnormalities were produced in the young by doses of 1000, 2000 and 3000 mg/kg (body weight). The maximal dosage at which no teratogenesis occurred was 500 mg/kg (body weight). The high dosages which were required to produce teratogenesis do not suggest any occupational hazard under normal conditions. As mentioned above, ETU is a breakdown product of a number of the ethylenebisdithiocarbamate fungicides and has been shown to cause birth defects in rats and mice. A study by Khera (1984) showed that ETU caused birth defects such as cleft palate and paw deformities when administered to Swiss-Webster mice in doses of 1600 mg/kg (body weight) and higher. However, at this dose, maternal toxicity was evident. Mice were seen to be about 50 times less sensitive than rats. The rate of formation of ETU in the field and the exposure of farm workers to this compound is likely to be low and in any case not much higher than in the general population. The hazard to farmworkers appears to be low.

Toxicity in Man

Use experience with maneb has been good. There are a few reports of contact dermatitis and allergic dermatitis (Shiaku, 1979: Arimatsu et al 1978).

Hazard Evaluation

It would appear that the hazard from the dithiocarbamate compounds is only evident at very high doses and that little effect can be expected in workers unless no precautions are taken. One possible side-effect is the interaction between some of these compounds (such as thiram) and alcohol.

Phthalimide Fungicides

This class of fungicides includes captan, captafol and folpet which together were used to an extent of about 120000 kg in 1983. They are all used as fungicides and as a class have a relatively low acute toxicity and have not been reported to cause problems in applicators except for allergic reactions (Hayes, 1982).

Captan

Captan is the most widely used chemical of this group in Ontario and is a fungicide which is used in fruit and vegetable production. It is available as a dust or wettable powder. The compound was originally tested by the U.S. company, IBT, for the purposes of registrations and has been the subject of recent controversy because some of the tests were incomplete. Both U.S. and Canadian registration authorities have requested new studies on the compound.

Environmental Factors

Captan is reasonably stable under environmental conditions except in the presence of alkali when it decomposes rapidly. The half life of captan in water at pH 8.5 (similar to most Ontario tap and well waters) is less than 30 min at room temperature and the material is almost completely removed from treated apples by washing and cooking (Frank et al 1983).

Absorption, Distribution, Metabolism and Excretion

Although rapidly absorbed through the skin, lung and stomach, the material is easily excreted. Within 24 hours of administration to rats, 90% of the captan was excreted and only 0.01 to 0.05% still remained in the organs (Seidler et al 1971). It has a short residence time in the body and does not accumulate. Studies on orchard workers (McJilton et al 1983) have suggested that the major route of contamination is via the skin. It is estimated that average skin exposure is 2.8 mg/person/hour while average respiratory exposure is 0.005 mg/person/hour. No health effects were observed under these exposure conditions.

Toxicity in Animals

Captan has an acute oral LD_{50} of 12-17 g/kg (body weight) in rats (Hayes, 1982), and is of low toxicity. In chronic studies, it seems to be quite toxic to cattle and sheep, with an acute oral LD_{50} of 250 mg/kg (body weight) in the former animal. Long term feeding trials in rats at 10000 ppm produced growth depression in both sexes and some atrophy of the testes. Dogs fed 100 mg/kg (body weight)/day for 48 weeks did not show significant pathological changes but at 300 mg/kg/day there were significant changes in organ weights. A number of tests have failed to show captan as either mutagenic in animals or teratogenic in animals treated at realistic doses (Hayes, 1982). However, a National Cancer Institute study (NCI, 1977) showed that mice treated at 8000 and 16000 ppm in the diet for 80 weeks developed cancer in the form of adenomatous polyps in the duodenum. Similar trials at 2200 and 6000 ppm levels in the diet did not cause the development of tumors. While the evidence that captan may produce cancer in mice is clear, this effect has not been obseved in man and may, at the high doses used in this study, have been due to artifacts such as gross overloading of the normal detoxification and excretion mechanism or the well-known gastric irritation effects of the material (Boyd and Carsky, 1971).

Effects on Reproduction

In a three generation study, rats reproduced normally when fed a diet of 1000 ppm (50 mg/kg (body weight)/day). In another study at doses of 2000 ppm no teratogenic effects were found but there was slight failure of growth in the fetuses. In rabbits fed 37.5, 75 and 150 mg/kg/day some malformation of fetuses occurred but these were not dose dependent (Kennedy et al 1968). In other studies captan was found to be non-teratogenic in rats, hamsters and two strains of rabbits. Other studies suggest possible teratogenesis in hamsters. In pregnant rhesus monkeys, captan did not have adverse effects on reproduction at 10, 25 or 75 mg/kg/day (Vondruska et al 1971).

Mutagenicity Tests

In general captan was mutagenic in bacterial screens but not in vivo.

Toxicity to Man

Use experience has been generally good. Few cases of occupational sensitivity have been reported, however, sensitivity was demonstrated in volunteers following application of the compound to their backs (Jordan and King, 1977). A study of chromosomes in workers employed in a captan factory failed to reveal any damage (Durham and Williams, 1972).

Hazard Evaluation

The evidence available to date suggests that prudent use of captan or other phthalimides does not present a measurable hazard to the farm worker. Its rapid breakdown under normal environmental and food preparation conditions suggests little concern for protracted exposure, bioaccumulation and possible long-term effects, however, its use just prior to consumption, such as for example in strawberries, should be avoided.

Phenoxy Compounds

This group of herbicides contains such compounds as 2,4-D, MCPA, dicamba, mecoprop, and MCPB. They represent a quite widely used group of pesticides with approximately 450000 kg used in Ontario on agricultural and roadside lands in 1983.

The mechanism of toxicity of phenoxy compounds in mammals is not fully understood, however, some salts of phenoxy inhibit oxidative phosphorlation and oxygen uptake in liver mitochondria and adversely affect peripheral nerve function. Phenoxy compounds are carried across the gut wall, lung and skin and are distributed in nearly all organs but show relatively higher concentrations in the kidney. They are not significantly stored in fat and are excreted in urine, usually within hours and at most days of exposure.

MCPA

MCPA is a phenoxy herbicide and is produced in three forms: MCPA ester which is marketed as an emulsifiable concentrate, and MCPA amine and the sodium and potassium salts which are available as aqueous solutions. It is used for the control of annual weeds in cereals, grassland and turf and is commonly used in mixtures of other herbicides.

Environmental Factors

MCPA is soluble in water and forms soluble salts of alkali metals and organic bases. MCPA is not very persistent in the environment.

Absorption, Distribution, Metabolism and Excretion

Absorption is by oral ingestion and inhalation. It is not readily absorbed through the skin. In rats administered MCPA by stomach tube,

absorption and elimination were rapid. The highest concentration in the blood occurred from 2 hours to 8 hours after administration and then diminished. During the first 24 hours, 92.3% of the dose was eliminated in the urine and 6-8% in feces (Elo, 1976). In human volunteers, 50% of the total dose was excreted in 48 hours, and five days following ingestion, no MCPA was detected.

Toxicity in Animals

The acute toxicity of MCPA is relatively low with the oral LD_{50} in rats and mice ranging from 550 mg/kg (body weight) to 1200 mg/kg (body weight). The intraperitoneal LD_{50} is reported as being about 300 mg/kg (body weight). In rat feeding studies over 7 months, levels of 400 ppm increased the relative weights of liver and kidney but did not produce histological changes (Gurd et al, 1965). High mortality, weight loss and histological changes in liver, spleen, kidneys and thymus were caused by daily dermal application of 1000 and 2000 mg/kg (body weight)/day.

Effects on Reproduction

There is no evidence that MCPA causes specific reproductive hazards or malformations but high doses can be toxic to the fetus.

Mutagenic Testing

MCPA is weakly mutagenic in some test systems.

Toxicity to Man

Most reported cases of poisoning in man have involved intentional or accidental poisoning rather than occupational hazards. At moderately low exposure MCPA can produce skin and eye irritation. In a factory in the U.S.S.R. it was considered to be the cause of contact dermatitis in

11% of 158 workers. At high exposure, the symptoms may include fever, headache, nausea, confusion, cardiac failure, kidney and liver injury and anemia may occur.

Hazard Evaluation

Most cases of acute poisoning with phenoxy compounds have been the result of attempted suicide or heavy exposure of manufacturing workers or applicators working without protection. In these cases, symptoms have varied from irritation of mouth, throat, and digestive tract, to abdominal pain and diarrhea. Absorbed phenoxy compounds have caused fibrillary muscle twitching, skeletal muscle tenderness, and myotonia (stiffness of the muscles of the extremities). Ingestion of very large amounts has produced metabolic acidosis, fever, tachycardia, hyperventilation, vasodilation and sweating (Morgan, 1982).

Some phenoxy herbicides may contain polychlorinated dibenzodioxins and dibenzofurans as impurities (Bainova, 1981). Although there are many polychlorinated dibenzodioxins, the one of greatest concern and toxicity is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). This compound is one of the most pharmacologically active in existence. Rhesus monkeys have proven highly susceptible to TCDD and one which received about 0.004 mg/kg (body weight)/day became allergic after 3 days and died on the 12th day. One which received 0.0004 mg/kg (body weight)/day died on the 76th day (McNulty, 1977). Rats following a dose of 0.2 mg/kg (body weight) died 40 days later. They showed no consistent appearance at autopsy, and the haematological changes they showed were probably associated with reduction of food intake (Grieg et al 1973). TCDD is also reported to be mutagenic, embryotoxic, teratogenic, and carcinogenic in experiments with different animal species (Bainova, 1981). The dioxin TCDD was only associated with the phenoxy pesticide 2,4,5-T, which is no longer used in Ontario, however, it was used in the past and some exposure may have occurred in the farming community.

The significance of this to the health and safety of the farming community is not known. The presence of other dioxin and dioxin-like contaminants in phenoxy herbicides is now regulated to 0.1 mg/kg of the technical material which is unlikely to present a hazard to users.

Bipyridyllium Compounds

The two common herbicides in this group, paraquat and diquat, were used to the extent of about 6500 kg in Ontario in 1983. These compounds are non-selective herbicides with low volatility and rapid inactivation under normal environmental conditions.

Of the two compounds, paraquat (Table 1-2) is the more toxic. A considerable amount of clinical experience has been reported in the literature. The chemical is relatively unique in the sense that it has an acute toxicity syndrome as well as the ability to produce a delayed fibrosis response in the lungs. The latter is usually the principal mechanism of death (Wagner, 1983). Most reported cases have been due to accidental ingestion and attempted suicides and few acute cases have been reported in agriculture workers. In acute poisoning, especially by the oral route, there may be irritation of the mouth and digestive system and renal and liver damage. Death may be caused by lung edema and heart failure. Lung symptoms do not usually occur until at least 3 days following ingestion. The fatal dose of paraquat is considered to be about 6 g or the equivalent of 30 ml of formulated product. Paraquat is one of the more toxic herbicides and, for this reason, its toxicity is highlighted below.

Paraquat

Paraquat is a post-emergent herbicide used in orchards and in the production of potatoes, asparagus and other crops. It is a quick acting herbicide which destroys green plants by contact action and

translocation and is effective in post-emergence weed control. It belongs to the chemical class of bipyridylliums and is sold in Ontario as an emulsifiable concentrate. It is known to cause fatalities in humans and other animals.

Environmental Factors

Paraquat is quickly rendered biologically inactive by absorption into clay minerals in the soil and is thus immobile.

Absorption, Destruction, Excretion and Metabolism

Paraquat is absorbed by the oral and dermal routes and by inhalation of droplets. Initially, the chemical, following intravenous injection in rats, is concentrated in kidney, lung and muscle at the same rate as in the plasma, but the rapid phase in the lungs ends after 20 minutes, compared with 1 to 4 hours in the other tissues. The lung becomes the organ of highest concentration after four hours and between that time and 10 days, paraquat concentration in the lung was 30 to 80 times that of plasma. The initial and secondary half-lives in plasma were 23 and 56 hours (Sharp et al 1972). When ^{14}C-labelled paraquat was given orally to rats, 25% of the radioactivity excreted in the feces could be attributed to products of metabolism by gut microflora. Urine from rats injected intraperitonealy contained 87% of the administered dose in 24 hours in the form of unchanged paraquat.

Toxicity to Animals

In rats, the oral acute LD_{50}s is 21 mg/kg (body weight) and the dermal LD_{50} 80-90 mg/kg (body weight). In guinea pigs oral LD_{50}s ranged from 20 to 60 mg/kg (body weight) and in rabbits is about 50 mg/kg (body weight). In monkeys, the LD_{50}s is 50-75 mg/kg (body weight). The principal effect appears to be damage to kidneys and adrenal glands.

Symptoms of vomiting, diarrhea, malaise and weight loss have been observed.

Four groups of eight A/He strain mice 9 to 15 weeks old were administered 50, 100, 200 or 300 ppm paraquat in drinking water for up to 16 weeks. Induction of smooth endoplasmic reticulum and the presence of lipidic lamellate cytosomes in the proximal convoluted tubule cells were observed in the kidneys of all paraquat treated animals (Fowler and Brooks, 1971). In order to investigate the carcinogenic potential of paraquat in mice, 4 groups of 70 males and 50 females received diets containing 0, 25, 50 and 75 ppm of paraquat ion for 80 weeks. All levels caused a slight to moderate reduction in body-weight increments, and these paralleled a reduced food intake. Twelve to 24 males and 12 to 23 females survived treatments for 80 weeks, the majority of deaths before this time being associated, in all groups, with respiratory disease and, in males, with the results of fighting. Thirty to 38 males and 31 to 38 females were subjected to histological examination. The incidence and types of tumours and other pathological changes in animals dying or killed at or before 80 weeks were similar in control and experimental groups (Fletcher et al 1972).

Effects on Reproduction

Groups of 12 male and 24 female rats were fed on diets containing 0, 30 or 100 ppm paraquat ion from 35 days of age. Three generations bred from these animals received the same diets during the whole period under test. Two litters were bred from each generation, and the effects on growth, food intake, fertility, fecundity, neonatal morbidity and mortality noted. No evidence was seen of damage to germ cell production or of structural and functional damage in these animals, and pregnant and young animals did not appear from this study to be more vulnerable to paraquat than adults. However, the incidence of renal hydropic degeneration in 3-4 weeks-old offspring was slightly increased in the 100 ppm group (Fletcher et al 1972).

Special Studies on Teratogenicity

On the second day after mating, groups of 10, 10, 6 and 5 female rabbits received, respectively, control diet, diet providing 1 mg/kg (body weight)/day paraquat ion, 2.4 mg/kg (body weight)/day paraquat ion for 8 days intravenously followed by 1.2 mg/kg (body weight)/day to term, or 1.2 mg/kg (body weight)/day paraquat intravenously for ten days followed by approximately 4 mg/kg (body weight)/day orally in drinking water to term. Offspring were examined for congenital abnormalities. Fertility and litter sizes were similar in the control and the orally dosed groups. Only one and three animals of the third and fourth groups respectively survived treatment.

Toxicity in Man

Paraquat is an irritant particularly to mucous membranes. Splashes of concentrate left in contact with skin cause irritation, inflammation and even blistering, and prolonged contact with nails leads to shedding. Contact with wounds delays healing. Inhalation of spray mist or dust will cause nosebleeds. Contact of solid with mucous membranes causes soreness, and splashes of liquid concentrate in the eye lead to severe inflammation which develops gradually, reaching its maximum after 12-24 hours. There may be extensive stripping of superficial areas of corneal and conjunctival epithelium and healing may be slow. Even in severe cases, effects of ingestion are due to the local irritant action: vomiting, abdominal discomfort and diarrhea and soreness of mouth and throat. Signs of severe kidney damage may appear in 2-3 days if large doses are absorbed. Large doses also cause tremors and convulsions. Signs of pulmonary injury may develop gradually after a few days, and these may lead on to breathing difficulties and pulmonary edema and fibrosis with death from respiratory insufficiency. The exact dose which is fatal to man is uncertain. The smallest dose known to cause death is 1 g, taken in the form of "Weedol" by a woman of 23. One man has

survived 3 g of paraquat, again as "Weedol" and one man recovered after swallowing 10 ml of 20% paraquat solution, despite being untreated for six days (Fisher, et al, 1971). From such evidence, the lethal dose in man would seem to be approximately 30 mg/kg (body weight). Estimates lower than this are based on accounts of subjects allegedly spitting out all of a dose, a process difficult to quantify.

For professional pesticide applicators, paraquat is not considered very dangerous if applied properly. Protective respiratory equipment is advisable in drifting spray, and dermal contamination especially of the hands, eyes and mouth should be avoided. Most human poisonings have resulted from accidents, suicides or criminal intent. Accidental intoxications have been the result of leaving paraquat within the reach of children or transferring the formulation to bottles used for beverages.

Hazard Evaluation

The occupational hazards of the bipyridillium herbicides are more inclined to include skin irritation, cracking of the skin, contact dermatitis and sometimes loss of fingernails. It is suggested that paraquat is neither mutagenic, carcinogenic nor teratogenic (Hayes, 1982). Diquat is much less toxic and less irritating to the skin and only a few minor accidents and occupational intoxications have been reported, but they were not severe. Diquat does not concentrate in the lungs as does paraquat, but lens cataracts have been produced in the eyes of experimentally treated animals. It would appear that, provided good precautions are taken, paraquat and diquat may be used with low hazard to farm workers.

Triazine Compounds

The most common herbicide in this group is atrazine, which is of moderate to low acute toxicity and its inclusion in this paper is mainly

due to its wide use in Ontario. 2.3 million kg of triazine herbicides were used in 1983. These compounds may persist in the soil for 1-2 years. They are only mildly irritating to skin, eyes, and upper respiratory tract, and systemic poisoning is unlikely unless very large amounts are ingested. Repeated dermal contact has, however, produced toxic dermatitis, skin sensitization and photosensitivity.

Atrazine

Atrazine is a triazine herbicide used for pre- and post-emergence weed control in the production of corn. It is sold in Canada as a wettable powder and a liquid concentrate.

Environmental Factors

Atrazine has moderate water solubility and volatility and is decomposed in the soil after 1 to 2 years. Frank and Sirons (1984) have reported a half-life in soils of 3-4 months under Ontario conditions.

Absorption, Distribution, Metabolism and Excretion

Following oral administration, technical atrazine and its metabolites were detected in the urine of pigs for slightly over 24 hours. The parent compound was the main material excreted. In addition, deethylatrazine was identified (Erickson et al 1978).

Chronic Toxicity

Atrazine was not tumorigenic when tested in mice at the highest tolerated dose which was 22 mg/kg (body weight)/day (Innes et al 1969).

Effects on Reproduction

High doses of atrazine (800 mg/kg (body weight)) given subcutaneously to pregnant rats produced some reabsorption of fetuses, but 200 mg/kg (body weight) produced no adverse effects. Atrazine has not been shown to produce fetal abnormalities in pregnant animals treated with atrazine.

Mutagenicity Testing

The compound was not mutagenic in yeast or Aspergillus screens and results in Drosophila melanogaster were inconclusive.

Toxicity to Man

A few reports of occupational exposure suggest that the compound may cause nausea and contact dermatitis which may be in the form of irritation, allergic reaction or photo-allergic response.

Toxicity to Animals

Oral LD_{50}s of 3000 and 1750 mg/kg (body weight) have been reported in rats and mice respectively (Bashmurin, 1974). The intraperitoneal LD_{50}s is 125 mg/kg (body weight) in rats. The symptoms of poisoning were excitation followed by respiratory depression, incoordination, and hypothermia. Death occurred 12 to 24 hours after oral administration and 4-6 hours after intraperitoneal injection.

A dermal dose of 2800 mg/kg (body weight) produced marked erythema but no systemic effects. The compound was irritating to the eyes (Gzhegotskiy et al, 1977).

Subacute Toxicity

No histological lesions were found in rats fed atrazine at a level of 100 and 500 ppm for 6 months, however some growth retardation occurred.

Hazard Evaluation

The hazard to the farming community from exposure to the triazine herbicides is considered to be low.

Anilide Compounds

The two commonly used herbicides in this group are alachlor and metolachlor. The total amount of this class used in Ontario in 1983 was about 1.9 million kg. These compounds are of low dermal and oral toxicity, but are mildly irritating to the skin, eyes and respiratory system. No occupational intoxications with these compounds have been reported, but work without protection could result in nerve, liver and haemoglobin disturbances and contact allergic dermatitis. Recently alachlor has been the subject of notice of cancellation by the EPA in the U.S.A. The reason for this is this result of recent tests which show that alachlor is a carcinogen. Current regulations in the U.S.A. state that alachlor must carry a label which warns of its carcinogenicity and its propensity to leach into surface and shallow groundwater aquifers.

Hazard Evaluation

The finding of alachlor and metolachlor in shallow wells in Ontario (Frank, personal communication) suggests a pathway of exposure for those persons who are dependent on water from these sources. This is of possible concern because of the large amount of alachlor used in Ontario. Recent reports also show that alachlor is carcinogenic in laboratory animals (Anon, 1984: Anon, 1984). Because of this,

Agriculture Canada is presently reconsidering the regulatory status of alachlor. The hazard to farmers who handle materials in which alachlor is present in concentrated form is probably high enough that rigid precautions should be taken to minimize exposure or an alternate material should be used until more information is available. At the present time, there is no evidence that metolachlor produces cancer in animals.

Urea Compounds

The common herbicides in this group are diuron, linuron and metabromuron. The amount of this class used in Ontario in 1983 was about 180000 kg. Some urea compounds are relatively stable in the environment and may persist for several years (Wagner, 1983).

Hazard Evaluation

The urea herbicides are of relatively low oral or dermal toxicity, but may cause mild to moderate irritation of skin, eyes, and respiratory system. In experimental animals these compounds have been found to cause weight loss, liver damage and deficiencies in the haemopoetic system at very high doses. The recent request from the EPA (EPA-OPP, 1984) for additional information regarding the reregistration of linuron suggests that this compound may represent a possible hazard. The EPA reports that the material has been shown to cause tumors in laboratory animals and has required that the manufacturer change the label to suggest additional protective clothing and precautions, particularly during the mixing and loading of the product (EPA-OPP, 1984). A cancer warning label is not required in the U.S.A. The regulatory status of linuron in Canada is currently under review. It seems appropriate that all precautions to minimize exposure to this compound be taken until further information is available.

Phenolic Compounds

Dinoseb

Dinoseb is a dinitrophenolic herbicide used as a contact and pre-emergence desiccant in the production of beans, peas, potatoes and vine crops. It is sold as an emulsifiable concentrate. It is considered a highly toxic herbicide and is readily absorbed through the skin.

Environmental Factors

This herbicide persists for about 2 weeks in the soil.

Absorption, Distribution, Metabolism and Excretion

The parent compound is metabolized by being enzymatically reduced in the liver to a variety of primary amines. Studies show that, following exposure, the chemical is found in the liver, kidney, spleen and blood (Wagner, 1983).

Toxicity in Animals

The oral toxicity in rats, mice and guinea pigs ranges from LD_{50}s of 20-46 mg/kg (body weight). The dermal LD_{50}s in guinea pigs is 200-300 mg/kg.

Information on chronic toxicity is sparse. A dietary level of 500 ppm caused marked refusal of food and death in rats after 5 days of administration, while 200 ppm caused an increase in blood urine nitrogen and increased weight of the liver. A dietary level of 50 ppm fed to rats for 6 monghs had no effect on growth, blood urea levels, organ weights or histopathological findings (Spencer et al, 1948).

In chronic feeding in ducks, cataracts were found after six months with a diet of 50 ppm. This syndrome has not been seen in other laboratory animals.

Effects on Reproduction

Dinoseb produced resorptions and reduced size of young when given to mice intraperitoneally or subcutaneously at the rate of 17.7 mg/kg (body weight)/day.

Toxicity in Man

Most reported cases of dinoseb toxicity in man have involved accidental ingestion of the chemical, however, Hayes (1982) has reported a case where a worker was exposed to dilute spray in one eye and developed pain and swelling of the eye. His vision was seriously impaired for three days. Recovery was complete.

In humans the symptoms are expected to be similar to those of other nitrophenols. These chemicals are toxic to the liver, kidney and nervous system and signs of poisoning include yellow staining of hair and skin, sweating, headache, lassitude, tachycardia, fever and convulsions.

Hazard Evaluation

This material has a high acute oral toxicity and should be used with caution, however, its effects in animals do not suggest a long-term hazard in man.

Discussion

The toxic effects of pesticides to farm workers are usually expressed either as an acute (immediate response to the compound) or as a chronic

effect. A number of pesticides show high acute toxicity and, even at low doses, cause easily recognizable effects from which rapid recovery usually occurs if the source of exposure is removed. These compounds, in general, are insecticides.

The organosphosphorus insecticides such as parathion, phorate, demeton, azinphos-methyl, disulfoton and terbufos all have high to moderate toxicity and, unless used in a careful and informed manner, may be hazardous by virtue of their acute effects. For the most part they have minimal long-term effects unless exposure is continuous. Continuous exposure is unlikely to occur in normal farming operations but may result from improper ventilation of pesticide storage areas.

Some of the carbamate insecticides also have high acute toxicity and possess a hazard profile which is similar to that of the organophosphorus compounds. Carbofuran, methomyl and aldicarb all have high acute toxicity and are hazardous if not used with appropriate precautions. Carbaryl is less acutely toxic. Chronic effects would not be expected to occur with carbamate insecticides under normal use conditions.

Certain pesticides, other than insecticides, may show high acute toxicity. For example, the highly toxic herbicides paraquat, diquat and dinoseb have a mechanism of toxicity which is common to both plants and animals. These compounds are normally, as in the case of insecticides, only hazardous if they are not handled in the correct manner.

Many other pesticides including certain insecticides, herbicides, fungicides and nematocides can be tolerated at quite high dose with little in the way of distinct symptoms of toxicity. This tends to breed a more casual attitude to the use and handling of these compounds and is of concern in the case of those compounds which may have chronic effects.

Some of these materials such as captan, alachlor, 1,3-dichloropropane and linuron have been shown to cause cancer in laboratory animals under certain conditions. In these laboratory tests, the pesticides are usually given to animals on a daily basis for most of their lifespan which is not typical of the pattern of farm worker exposure. In some cases, (i.e. captan) these compounds cause cancer only at high doses which would not be expected to occur during normal farm operations. In other cases the compound may be given via a route by which exposure would not normally occur (i.e. 1,3-dichloropropanes given via a stomach tube). The significance of cancer induced in animals under laboratory test conditions to the health of farm workers is unclear. These compounds are under active review in the U.S.A. and Canada and, until more information is available, they should be treated with the care and caution normally associated with compounds of higher toxicity.

A problem in the evaluation of the toxicity of any substance, including pesticides, is that many of the studies have to be carried out in laboratory animals. This raises the inevitable question of the relevance of observations made in animals which may not have an exactly similar physiology or biochemistry to that of man. A lack of toxicological procedures which better address the hazard of pesticides to Ontario farm workers is noted.

Incidence of Pesticide Poisoning

Health and Welfare Canada compiles data on poisoning on a year by year basis for the whole population and presents this data by province and by type of poison. Table 3-2 shows the number of reported poisonings (due to all poisons) in Canada, in both rural and urban dwellers, those that required treatment or hospitalization and those that resulted in mortality. The poisonings are also listed in three age groups representing small children/toddlers, older children and adults. It is relevant that, for total poisonings, children under the age of 4 years

were most often involved. From the data on poisonings reported to have occurred from exposure to pesticides (Table 3-3) in both Ontario and Canada, the proportion of young children involved and the number of deaths were lower than might be expected. The data presented in these tables gives the impression that Ontario has the majority of lethal poisonings, however, this is a result of the better data collection system for this province. Only Ontario includes coroner's death reports in poisoning statistics; the other provinces do not, and cases are missed for this reason. The number of lethal pesticide poisonings in Canada is, in fact, higher than indicated here. For example, Statistics Canada's mortality data for 1979 and 1980 listed 15 and 12 poisoning deaths due to pesticides respectively while the data collected from the poison control centres listed only 5 and 3 respectively. In some years deaths from pesticide poisoning were mostly associated with suicide. The statistics currently available suggest that death due to poisoning by pesticides is an unlikely event, especially in Ontario. However, the number of non-fatal cases of poisoning was high and, as the data were collected from reports to poison control centres, may be underestimates of actual incidence in the general population. Also of interest is the apparent increase in the number of reported poisonings in persons older than 15 which has been observed in the last 5 years. It is uncertain whether this is a change in reporting or an actual change in cases.

Table 3-2

Total Poisoning Cases In Canada, 1979-1983

YEAR	AGE GROUP IN YEARS				PHONE CALLS	TREATED OR HOSPITALIZED				DEATHS		
	TOTAL	0-4	5-14	15+		TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
1979	58028	36967	4566	10979	26615	13569	6620	1222	5268	166	33	113
1980	64822	41982	5079	12296	30188	23178	12446	1829	7368	222	37	178
1981	71519	45252	6330	17599	40501	3642	978	318	2238	250	43	193
1982	80807	51880	6614	19435	50998	29809	15460	2316	11036	255	45	190
1983	83995	54933	6986	19814	52901	31094	16490	2363	11375	234	48	173

* The unusual decrease in treated poisonings in 1981 is due to a change in the method by which these were reported. The numbers in the total column are, in some cases, not equal to the sum of the numbers in other columns. The reason for this is uncertain, the data are presented as given.

Source: Health and Welfare Canada.

Table 3-3
Pesticide Poisonings In Canada and Ontario, 1979-1983

YEAR		AGE GROUP IN YEARS				PHONE CALLS	TREATED OR HOSPITALIZED				DEATHS		
		TOTAL	0-4	5-14	15+		TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
CANADA	1979	2182	1383	177	238	1179	279	228	23	22	5	0	5
ONT.	1979	869	552	84	51	737	97	85	6	5	4	0	4
CANADA	1980	2493	1587	204	346	1040	839	554	62	135	3	0	3
ONT.	1980	1034	661	88	71	408	265	163	25	24	1	0	1
CANADA	1981	2952	1822	282	682	1494	97	55	4	33	5	2	2
ONT.	1981	1249	777	143	294	659	36	23	1	11	1	1	0
CANADA	1982	3224	1966	282	767	1958	1266	807	94	310	5	2	1
ONT.	1982	1403	871	129	349	947	456	277	37	125	5	2	1
CANADA	1983	3404	2093	291	876	1946	1458	928	102	376	2	0	3
ONT.	1983	1521	918	151	415	960	561	327	47	171	2	0	3

* The unusual decrease in treated poisonings in 1981 is due to a change in the method by which these were reported. The numbers in the total column are, in some cases, not equal to the sum of the numbers in other columns. The reason for this is uncertain, the data are presented as given.

Source: Health and Welfare Canada.

Table 3-4

Pesticide Poisoning in Canada in 1983 by Agent

	AGE GROUP IN YEARS			PHONE CALLS	TREATED			DEATHS		
	TOTAL	0-4	5-14	15+	TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
Algae control	7	7	0	0	3	4	4	0	0	0	0
Algaecide	13	7	2	4	6	7	1	2	4	0	0	0?
Ant poison	95	77	6	10	62	33	25	2	5	0	0	0?
Arsenic	6	0	0	6	3	3	0	0	3	0	0	0
Black Flag Ant Trap	23	20	1	0	14	9	7	0	0	0	0	0
Black Flag Insect.	11	3	1	7	7	4	0	0	4	0	0	0
Borax	9	7	0	1	6	3	3	0	0	0	0	0
Boric Acid	19	8	2	9	11	8	2	2	4	0	0	0
Captan	52	30	12	6	20	32	14	9	6	0	0	0
Chipman Insecticide	6	0	1	4	4	2	0	0	2	0	0	0
Chlordane	71	27	4	32	37	34	14	1	17	0	0	0
Citronella Ins. Repel.	7	5	0	2	2	5	4	0	1	0	0	0
Cockroach Poison	26	4	3	17	21	5	1	1	3	0	0	0
Creosote	70	23	9	37	49	21	12	1	8	0	0	0
Creosote (creolin)	1	0	0	1	0	1	0	0	1	1	0	1
Creosote wood preser.	7	5	1	1	6	1	0	1	0	0	0	0
Cygon insecticide	48	7	2	34	26	22	3	1	16	1	0	1
Diazinon	159	47	14	83	115	44	13	3	24	0	0	0
Dursban insecticide	16	4	1	11	13	3	0	0	3	0	0	0
Fertabs plant food	37	32	2	2	27	10	9	0	0	0	0	0
Fertilizer	180	137	14	23	147	33	22	2	9	0	0	0
Flea collar	14	12	1	1	14	0	0	0	0	0	0	0
Flea powder	10	5	2	3	9	1	0	0	1	0	0	0

	AGE GROUP IN YEARS			PHONE CALLS	TREATED				DEATHS		
	TOTAL	0-4	5-14	15+	TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
Flea shampoo	6	5	0	1	5	1	1	0	0	0	0	0
Flea spray	7	2	0	5	6	1	1	0	0	0	0	0
Fossil flower insect.	7	1	0	6	7	0	0	0	0	0	0	0
Fungicide	14	5	3	5	8	6	1	2	2	0	0	0
Furadan insecticide	24	3	0	19	4	20	2	0	17	0	0	0
Gramoxone insecticide	6	1	1	4	3	3	0	0	3	0	0	0
Green Cross Ant Kill.	48	41	2	3	32	16	14	0	1	0	0	0
Green Cross Insect.	19	10	0	9	11	8	3	0	5	0	0	0
Green Cross Mouse Poison	13	13	0	0	9	4	4	0	0	0	0	0
Green Cross Rose Dust	5	1	0	3	2	3	1	0	1	0	0	0
Green Cross Wasp & Hornet Blaster	5	1	0	5	5	0	0	0	0	0	0	0
Herbicide	28	7	7	14	21	7	2	1	4	0	0	0?
Insect	18	11	1	6	18	0	0	0	0	0	0	0?
Insect repellent	39	32	2	4	27	22	17	1	4	0	0	0
Insecticide	133	62	7	54	90	43	18	1	20	1	0	1?
Kerigard insecticide	7	7	0	0	5	2	2	0	0	0	0	0
Killer Cane Herbicide	5	5	0	0	4	1	1	0	0	0	0	0
Killex Herbicide	50	26	4	17	30	20	10	0	10	0	0	0
Laters insecticide	17	8	0	8	13	4	3	0	1	0	0	0
Lindane insecticide	9	4	1	4	1	8	3	1	4	0	0	0
Malathion	113	24	8	68	72	41	8	2	27	0	0	0

	AGE GROUP IN YEARS			PHONE CALLS	TREATED			DEATHS		
	TOTAL	0-4	5-14	15+	TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
Marquette insecticide	6	3	0	3	4	2	1	0	1	0	0	0
Mosquito Coil	18	14	1	2	10	8	7	0	0	0	0	0
Mouse Bait	24	14	8	2	13	11	8	3	0	0	0	0
Mouse Poison	52	42	5	4	23	29	23	2	3	0	0	0
Mouse Poison Warfarin	11	9	1	0	5	6	5	0	0	0	0	0
Mouse Poison Seeds	6	6	0	0	1	5	5	0	0	0	0	0
Mouse Treat	26	26	0	0	9	17	17	0	0	0	0	0
Muskol Insect Repel.	135	115	6	7	71	62	52	2	5	0	0	0
Noxzema Insect Repellent	8	8	0	0	4	4	4	0	0	0	0	0
Off Insect Repellent	113	86	19	5	77	36	27	6	3	0	0	0
Organophosphate Insecticide	9	1	0	8	0	9	1	0	8	0	0	0
Ortho Insecticide	16	9	7	0	15	1	0	1	0	0	0	0
Paradichloro-benzene	36	32	2	2	26	10	9	0	1	0	0	0
Paraquat Herbicide	16	1	1	14	10	6	1	1	4	0	0	0
PCP	26	2	1	18	4	22	2	1	15	0	0	0
Pentachloro-phenol	9	3	1	5	5	4	3	0	1	0	0	0
Pentox Wood Preserv.	17	4	4	9	7	10	3	1	6	0	0	0
Pesticide	18	5	1	9	14	4	0	1	3	0	0	0
PIC Coil	10	9	1	0	7	3	3	0	0	0	0	0
Polyram 7 Fungicide	5	5	0	0	0	5	5	0	0	0	0	0
Propoxur	6	1	0	5	4	2	1	0	1	0	0	0

	AGE GROUP IN YEARS			PHONE CALLS	TREATED			DEATHS		
	TOTAL	0-4	5-14	15+	TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
Raid Insecticide	110	24	16	66	82	28	2	3	23	0	0	0
Rat Poison	167	127	17	22	66	101	78	10	13	0	0	0
Rat Poison-Warfarin	44	34	3	7	17	27	20	1	6	0	0	0
Ratak Rat Poison	23	20	2	1	8	15	15	0	0	0	0	0
Roach Killer	5	4	0	1	3	2	2	0	0	0	0	0
Rodenticide	5	5	0	0	4	1	1	0	0	0	0	0
Rotenone	7	3	0	4	5	2	1	0	1	0	0	0
Round-up Herbicide	11	1	2	7	5	6	1	1	3	0	0	0
Scent-Off Animal Repellent	5	5	0	0	3	2	2	0	0	0	0	0
Sevin Insecticide	30	10	5	14	20	10	3	2	5	0	0	0
Slug Bait	5	5	0	0	4	1	1	0	0	0	0	0
Sorexa Mouse Bait	12	10	1	1	6	6	5	0	1	0	0	0
Sting-Bee	121	35	32	42	109	12	1	10	1	0	0	0?
Sting-Hornet	14	3	1	8	12	2	1	0	1	0	0	0?
Sting-Insect	8	1	3	4	7	1	0	1	0	0	0	0?
Sting-Unknown	6	3	0	2	6	0	0	0	0	0	0	0?
Sting-Wasp	116	25	23	64	108	8	2	2	4	0	0	0
Temik Insecticide	6	0	0	6	1	5	0	0	5	0	0	0
Thiram	5	3	0	1	3	2	1	0	1	0	0	0
Treated Seeds	17	16	1	0	10	7	7	0	0	0	0	0
Warfarin	49	36	4	8	23	26	21	2	3	0	0	0
Weed & Feed	14	6	0	8	12	2	1	0	1	0	0	0
Weed Killer	17	6	4	4	13	4	1	1	2	0	0	0?
Weedex Herbicide	6	4	2	0	4	2	2	0	0	0	0	0

	AGE GROUP IN YEARS				PHONE CALLS	TREATED			DEATHS		
	TOTAL	0-4	5-14	15+	TOTAL	0-4	5-15	15+	TOTAL	ACDT.	SUIC.
Wilsons Insecticide	28	11	3	13	18	10	4	1	4	0	0	0
Wilsons Mouse Treat	25	23	0	2	9	16	14	0	2	0	0	0
Yellow Jackets (Std)	7	1	0	5	1	6	0	0	5	1	1	0?
2-4-D	37	10	7	17	24	13	4	2	5	0	0	0
6-12 Insect Repel.	14	13	1	0	9	5	5	0	0	0	0	0

* Those entries followed by a question mark are considered questionable for lack of clarity or overlap with other classes. The numbers in the total column are, in some cases, not equal to the sum of the numbers in other columns. The reason for this is uncertain, the data are presented as given.

Source: Health and Welfare Canada.

The data in Table 3-4 show the range of chemical substances which were involved in pesticide poisonings in 1983. Again, the lack of specificity in the diagnosis and the obvious overlap of categories and classes suggests that the reporting system should be revised.

It is important to point out that the data available from Statistics Canada do not allow identification of farm related poisoning. However, California has an injury reporting system operated under the California Workman's Compensation Law and which allows the Department of Agriculture and Food access to the reports as well as allowing further investigations to be carried out. This system results in about 85% reporting of injuries but does not include those not covered by workman's compensation, i.e. the farmer, unpaid family labour, self-employed operators and field workers who are not U.S. citizens. In comparison to the situation in California, the Workers' Compensation Board of Ontario reported only 7 cases of pesticide related poisoning in 1983 (WCB, 1983). Of these, one was reported from a farmworker while the rest were reported in occupations ranging from nursing aid to sewing machine operators. Bearing in mind the similarity in the size of the population of California and Canada, the greater use of pesticides in California than any other U.S. state and the differences in climate, both from the point of view of the types of pests present and the type of field clothing worn by workers, it seems likely that pesticide exposure in Canada, and in Ontario, will be less than that experienced in California. The pesticide-related injuries reported in California are, however, worthy of study because they do allow an estimation of the hazard associated with different operations on the farm and may point to areas of greatest potential concern here in Ontario.

Injuries leading to disability (inability to report for the next shift) in California were surveyed between 1965 and 1971 by Whiting (1975). The number of employees in the agricultural sector is greater than in the industrial sector and he showed that, although the number of injuries in

the agricultural area was high at 5% of the workforce, the number of fatalities was low at 59 compared to the 711 reported for the industrial sector. Almost all the fatalities were caused by motor vehicles and the deaths due to poisoning by agricultural chemicals varied between 0 and 3 per year. A study of the pesticide related illness cases showed that these occurred more frequently in ground applicators, mixers, loaders and field workers than in other occupations. Illness in bystanders due to pesticide spray drift, and in flaggers and aerial applicators was lowest of all.

Discussion

The published information on both fatal and non-fatal poisoning in Ontario farmers is inadequate in that it does not provide a comprehensive analysis of the incidence, geographic location, occupation and severity of pesticide poisoning in Ontario.

Non-specific Health Effects of Pesticides

In addition to the systemic effects of pesticides, various types of dermatitis have been well documented in man. Some types of dermatitis may affect, in a general way, most workers who are exposed to certain direct irritants while allergic responses may occur only in sensitized subjects who show stereotyped symptoms in which immunological changes are associated with exposure to the chemical.

Contact Toxic Dermatitis

Recent studies have shown that 10% of all types of dermatitis are work-related and that 80% of these cases are caused by skin irritants.

Pesticides may be primary irritants, e.g. phenoxy herbicides and anilides, or delayed irritants, e.g. bipyridillium herbicides. Most herbicides and fungicides and some insecticides act as skin irritants even to the degree of producing gross blisters as in the case of methyl bromide.

Contact Allergic Dermatitis

This is a delayed type of skin sensitization produced by many pesticides, e.g., some organophosphorus compounds, urea compounds, dithiocarbamates, triazines, amines and benzimidazoles. The sensitizing agent does not cause visible changes in the skin after the first contact, but after several months, allergic symptoms occur at each contact with the offending chemical. Contact allergic dermatitis does not occur among many exposed workers, and this condition is found more frequently among greenhouse workers than among those working in the field. The allergic eruptions vary in severity but many result in acute or chronic eczema.

Epidemiological or case reports suggest that the following products are contact allergens: benomyl, dinoseb, lindane, malathion, simazine, trifonate and zineb.

A further modification of allergic dermatitis is photo-allergic dermatitis. This condition is caused by exposure to photosensitizing pesticides, with excess of sunlight acting as a provoking factor. Anilides, dithiocarbamates, triazines and urea compounds are known to be responsible for several cases of photocontact dermatitis among agricultural workers. Formulations of importance are atrazine, benomyl, simazine, thiram and zineb.

The well-known effect of exposure to sublethal levels of organophosphorus insecticides is a lowering of the serum and red blood cell cholinesterase levels but Levin et al (1976) also reported that

similar exposure could cause increased levels of anxiety. This study only showed this effect in persons who used organophosphorus pesticides on a daily basis. Farmers who also used organophosphorus compounds were apparently not affected, although the sample was small.

In many cases, the symptoms of pesticide poisoning may be mistaken for other illness such as viral disease or hangover (Whiting, 1975) or, as reported by Leftwich et al (1982), a case of undiagnosed fever which was found to have been caused by exposure to herbicides containing 2,4-dinitrophenol compounds during cleaning of a spray tank.

Chlorothalonil has been reported to cause allergic reactions in skin of exposed woodworkers (Johnsson et al 1983). Palva et al (1975) reported a case of aplastic anaemia from skin exposure to a spray solution of MCPA contained in a leaking sprayer. Similar contact toxicity was reported for paraquat (Levin et al 1979) where skin contamination from a leaking sprayer caused the death of one worker and reduced lung function in several others. Paraquat is highly toxic when consumed (in error or intentionally) but was not considered hazardous by skin absorption until recently.

CHAPTER FOUR
TOXIC EFFECTS OF OTHER FARM CHEMICALS

Dusts and Other Particulates

Farmer's lung has long been recognized as an occupational disease in a number of countries (Hapke et al 1968). This disease of the lungs and respiratory system is caused by inhalation of dust from mouldy hay and other substances. It may present itself as a series of recurring episodes, continuous illness or solitary episodes. In some cases, the condition is only temporary but in others it may cause mild to total disability. The clinical features of the disease are a result of fibrosis of the lungs which leads to decreased pulmonary function and difficulty in breathing. Fibrosis is thought to be caused by allergic reaction to inhaled spores and other fungal products and a number of fungi have been identified as possible causative agents. Hapke et al (1968) reported the involvement of Thermopolyspora polyspora, Aspergillus fumigatus and Actinomyces spp. Imbriani and Catenacci (1980) reported the involvement of Micropolyspora faeni and Thermoactinomyces vulgaris and Roberts et al (1983) reported the involvement of T. candidus. Braun et al (1983) have, however, pointed out the presence of anti-lung antibodies in farmers showing the symptoms of farmer's lung and have suggested that these may play a part in the expression of the condition. A report by Kay (1974) suggested that silica particles may play an important role in conditions such as farmer's lung and pointed out that many pesticides are formulated with silica containing carriers. Later studies by Sherwin et al (1979), using sophisticated electron microscopy and energy dispersive x-ray analysis allowed the identification of silica particles in the lungs of farm workers from California. Collection of soil samples revealed the existence of similar particles. Although it is likely that silicates from the soil are responsible for these types of conditions, clays and silicates in pesticide formulations may be responsible and extra precautions may be required when using these formulations of pesticides.

The epidemiological studies which have been carried out report a significant incidence of farmer's lung. Madsen et al (1976) reported an incidence of 14% in a survey of 471 farmers in Wyoming while Babbott et al (1980) reported an incidence of 6 to 18% in white, male, dairy-farmers from Vermont. A study of Manitoba farmers (Warren, 1981) has shown that 50% of a sample of 76 persons showed respiratory problems associated with the handling of crops. As pointed out by Warren (1980) and extensively reviewed in Dosman and Cotton (1980), exposure to grain dust has been recognized as a health hazard in Canada and in other countries (Kavoussi, 1974) for some time. Clinical studies (Cotton et al, 1982) on cereal grain workers in Saskatchewan grain elevators showed a synergistic effect of smoking and exposure to grain dust on lung dysfunction. This study did not identify fungal spores or allergic response to fungal products as a cause of the condition. Broder and McAvoy (1981) have demonstrated an immunological reaction between human serum and grain dust. The response was also seen with crushed clean grain kernels which suggests that it is an intrinsic property of the grain and is not a contaminant such as a pesticide. A number of studies conducted in Europe have reported an association between barn- and farm-associated respiratory allergies and the mites which are found in hay (Ingram et al, 1979: Cuthbert et al 1979: Cuthbert et al 1980: Hillerdal et al 1982: Terho et al 1982). This condition seems to be more prevalent in the cooler climate of North Europe where cattle, feed and farmers spend many months of the year in barns. This suggests that a similar problem may occur here in Ontario. Although farmer's lung is normally associated with persons who have had a long exposure to farming, Bureau et al (1979) reported a case in a five-year-old boy from Quebec. Conditions similar to farmer's lung have been reported in mushroom workers (Stewart, 1974) and compost plant workers (Vincken and Roels, 1984). Mushroom worker's lung has been associated with the growth of Aspergillus fumigatus and Humicola grisea in spent compost (Kleyn et al 1981).

The presence of the fungi in dust can be demonstrated with the aid of microbiological techniques (Treuhaft and Arden Jones, 1982) but this does not prove cause and effect. Identification of the condition can sometimes be made with the aid of immunological responses to the fungi identified as causative agents in the disease (Katila and Mantyjarvi, 1978: Berrens et al 1977: Patterson et al 1976: Mantyjarvi et al 1980: Schorlemmer et al 1977), however, other workers have reported a poor correlation between immune response to fungal antigens and the condition (Marx et al 1978: Edwards and Davies, 1981). Turton et al (1981) suggested the use of enzyme markers such as serum lysozyme as aids in the diagnosis of the condition.

Other environmental exposure to hazardous substances may also occur in the farming environment. Veien et al (1980) reported contact dermatitis in farm workers exposed to the therapeutic antibiotics, spiramycin and tylosin, in Danish farmers and van Ketel and van Diggelen (1982) reported a dairy farmer who was allergic to cows. Respiratory symptoms in farmers harvesting grain have been shown to be associated with allergic response to the spores of fungi associated with the grain in the field. Cladosporium spores were the most abundant and could reach levels of 200 million/m^3 near the combine and 20 million/m^3 near the driver (Darke et al 1976). Dutkiewicz (1978a and 1978b) reported the presence of large numbers of bacteria in farm and grain dust. Bacteria which were identified were Erwinia herbicola, Staphylococcus, Corynebacteria and Streptococci in order of predominance. Grain handlers showed immunological reactions to Erwinia herbicola, suggesting a link to respiratory disorders. Thedell et al (1980) reported the presence of endotoxins from gram-negative bacteria in the air in hog and poultry houses in the American Midwest, although no adverse health effects were noted in the workers in these areas. Matson et al (1983) were unable to show allergy to hog proteins in a sample of 35 hog-barn-allergic farmers in Minnesota. Similar results were reported in studies on hog producers from Finland although these farmers were shown to be

allergic to substances found in the feed given to the hogs (Katila et al 1981). A survey of thirty hog producers in Iowa (Donham et al, 1977) showed high concentrations of dusts in the buildings. Studies on a number of Southern Ontario hog producers (Nethercott et al 1983) showed nasal and eye irritation associated with hog production and 60% (out of 50 farmers) showed allergic skin reactions to grain dust while only 20% showed allergic reaction to hog dander.

Burg et al (1981) have reported finding aflatoxin B_1 in dust samples collected during the handling of contaminated corn in the Midwestern U.S.A. Concentrations ranged from 1 to 42 ug/kg in the dust and most was associated with the larger ($> 7\mu m$) dust particles. Despite the low concentration found, the authors draw attention to the very high toxicity of the aflatoxins and suggest that suitable precautions be taken in the handling of contaminated produce. Gerberick and Sorenson (1983) have shown that T-2 toxin, a fungal toxin produced by several Fusarium spp, (one of the so-called trichothecene group) has a significant cytotoxic effect on lung tissues in laboratory studies. Field effects of this toxin have not been reported in farmers but the fungus and the toxin are well known from several small grain crops.

Exposure of farmers to dusts and particulate matter from insulation materials such as asbestos and glass-fiber may also have hazardous effects on health. Although much information is available with respect to occupational exposure to these materials, there is none which deals specifically with the farm as a source and the farm worker as the recipient. There is no reason to believe that the farm worker is at any greater risk than other persons such as construction workers who are exposed to these materials during the course of their occupation.

Apart from the explosive nature of some dusts (not addressed in this document), it is obvious that some hazard is associated with the handling of agricultural produce which is dusty and/or contaminated with fungal

spores, fungal or bacterial antigens or toxins. In most cases the condition is not lethal and can be treated with suitable drugs, however, prevention of exposure is also effective and can be carried out as simply as switching to silage instead of hay (if justified by other factors such as cost or feasibility) or by the wearing of a good respirator while working in dusty areas (Cuthbert and Gordon, 1983).

Gases

A number of gases are used and produced during farming operations. Ammonia, as an anhydrous gas, is used as a fertilizer by direct injection into the soil. Exposure may occur during this operation and could result in serious or even lethal effects. Precautions, such as the use of a respirator and the strong irritating and therefore warning effect of ammonia allow its relatively safe use in farming operations.

More hazardous are those gases which are produced on the farm and which may collect in structures. These are: methane, hydrogen sulphide and carbon dioxide, produced from the fermentation and rotting of organic matter; ammonia, produced from the decomposition of nitrogen-rich waste and nitrogen dioxide, a gas formed in grain silos.

A survey of thirty hog producers in Iowa (Donham et al, 1977) showed high concentrations of toxic gases in the buildings. In all cases levels of carbon monoxide, carbon dioxide, ammonia and hydrogen sulphide were higher in winter than in summer and in all cases TLV limits were exceeded in one or more of the buildings. In a study of six hog production units in Southern Ontario (O'Blenis et al 1983) dust and ammonia levels were high enough to produce chronic respiratory problems in workers. Concentrations of other gases were not measured. They also recommended several mechanical and protective strategies for reduction of exposure.

Silo-fillers disease is a rapidly appearing cough and respiratory difficulty syndrome which is caused by nitrogen dioxide or silo gas. This gas is produced by the oxidation of nitrates in crops grown in nitrogen rich soils (Warren, 1977). The hazard is highest during the filling of the silo since the gas will disperse after several weeks. The recent death of two farm workers in Northern Ontario (Bennett, personal communication) from this gas suggests that it is an important hazard.

The problem of noxious gases is difficult to solve but increased ventilation through air-to-air heat exchangers in barns may offer a solution in winter conditions. The problem of silo gas is more difficult to solve but information on silo gas is available and adherence to normal safety precautions and education programs could be beneficial. These and other educational suggestions were offered by the coroner's jury in the above case (Bennett, personal communication). Some of these issues were recently addressed (November, 1984) at a conference in Kitchener. This suggests an increased awareness of the problem, although the conference proceedings have not yet been published and are thus not generally available.

Mycotoxins

In addition to the production of spores, which are involved in the respiratory diseases already described, fungi may grow on field crops and in stored cereals and corn, producing metabolic products which may be toxic to man and animals. These metabolic products are called mycotoxins. Although the main concern is directed to the effects on the consumer, be it man or animal, of ingestion of these contaminants in food, there is a possibility of absorption by inhalation and dermal exposure in those handling grain products.

There are many mycotoxins but those of current importance in Canada include zearalenone and the tricothecenes. Both of these have been found in most parts of Canada.

Zearalenone

Zearalenone is a non-steroidal estrogenic mycotoxin produced by fungi of the genus Fusarium. Outbreaks of disease due to the presence of this mycotoxin in pig and cattle feed have been reported in many countries including the United States and Canada. The disease is basically one of estrogenicity and the syndrome is characterized by fetal death, infertility, reduced litter size and vulvovaginitis in females and feminizing effects in males including testicular atrophy and mammary gland enlargement. There is no clear evidence that zearalenone is teratogenic, results in animal experiments being equivocal (Ruddick et al 1976). There is no direct evidence that this mycotoxin is carcinogenic, although in long term studies in B6C3F mice zearalenone increased the number of pituitary adenomas and hepatocellular adenomas. This did not occur in F344/N rats.

Trichothecenes

The trichothecenes consist of about 60 mycotoxins produced by many genera of fungi including Fusarium, Myrothecium, Trichoderma, Cephalosporium and Stachybotrys. These mycotoxins include T-2, crotocin, macrocyclic and nivalenol type toxins. Of these, the most important in Canada is deoxynivalenol (Vomitoxin), a contaminant of corn, barley and wheat.

Poisoning by these toxins occurs in cattle, swine, horses, poultry and, in Asia, in man. These toxins have an affinity for dividing cells in various organs of the body and cause necrotic lesions. Although the syndrome is variable, inflammation and necrosis of the upper alimentary tract from the mouth to the stomach is characteristic. In addition, atrophy of bone marrow and haemorrhage in various organs may occur. The symptoms of poisoning include reduction of feed intake, vomiting, tremors and convulsions. Ueno (1984) has reported that the T-2 toxin and the

macrocyclic trichothecenes cause dermal irritation, haemorrhage of the intestines and destruction of the thymus in experimental animals. The other toxins also produced similar results but at much higher doses. No skin tumor formation was noted in the case of fusarenone-X, one of the macrocyclic toxins, suggesting that these compounds are not carcinogenic. Khera et al (1984) studied the effects of vomitoxin on reproduction in mice and rats. At concentrations of up to the equivalent of 2 mg/kg (body weight)/day the material had no effects on reproduction although it did have an effect on both survival and weight gain of the pups from treated mothers.

Discussion

Dusts, particulate matter, mycotoxins and gases present a significant health hazard to the farm worker. It is noted that there is an apparent lack of awareness of these hazards and a lack of use of already available respiratory protective equipment. Little attention appears to have been given to the design of structures and equipment that would minimize exposure to these hazards.

CHAPTER FIVE

EPIDEMIOLOGICAL INVESTIGATIONS ON FARM WORKERS

A number of epidemiological studies have been carried out in farm workers and these are discussed in the sections below.

Pesticides and Cancer in the Farming Community

Cancer is very much a part of both life and death in Canada and Ontario and its effects are seen in the urban as well as the farming community. A number of studies have been conducted in which farming as a profession has been associated with higher than normal rates of cancer. It must be emphasized that these increased cancer rates, when reported, are not necessarily related to pesticide exposure. In fact, no epidemiological studies are available that demonstrate a clear link between cancer and exposure to pesticides. No studies of this relationship have been made in Ontario, therefore, this review will summarize data from the world literature.

Burmeister et al (1983) showed in an analysis of death certificates of residents of Iowa dying between 1964 and 1978 that a number of cancers were more prevalent in farmers than in a control population. Deaths from multiple myeloma, lymphoma, prostate and stomach cancer were all higher than would be expected. These higher incidences of multiple myeloma were associated with egg and hog production as well as with counties having high herbicide and insecticide use. However, it was not possible to relate individual use of pesticides or individual farming practice with increased rates of cancer. Buesching (1984) conducted a similar analysis of death certificate data in Illinois and showed that male farmers in Winnebago County had increased rates of non-Hodgkins lymphoma and prostate cancer, although lung and stomach cancer were lower than expected. It was not possible to associate these increases

with pesticide use, although pesticides were more widely used in this county than in a control county. Blair and White (1981) conducted a death certificate study of Wisconsin farmers based on records collected from 1968 to 1976 but did not show a statistically significant increase in incidence of leukemia in the whole farming population. However, on a county basis it was possible to observe increased risk of leukemia associated with dairy production and high fertilizer use. Cantor (1982) observed death certificate data for farmers in Wisconsin dying of non-Hodgkin's lymphoma between the years of 1968-1976 and noted an increased risk of cancer associated with farming. In a county-by-county analysis, it was shown that risk of lymphoma increased with increasing agricultural activity in general, increased planting of small grains, wheat and increased use of insecticides. Cantor and Blair (1984), in a study of multiple myeloma in Wisconsin based on information from death reports, found that farmers surveyed from 1968 to 1976 had a higher than normal chance (as determined from the odds ratio) of dying from multiple myeloma. Their study showed that 26.8% of farmers died of multiple myeloma as compared to 21.7% of a control population matched for age, time of death, marital status etc. Similar but slightly higher incidences were noted in farmers older than 65 years at time of death, as well as in farmers from counties of high chicken production, fertilizer or insecticide use. In most cases the confidence intervals in these observations were large and the sample of farmers observed was rather small at 110 persons. In common with many other studies, it was not possible to determine whether a farmer from a county of high pesticide or fertilizer use would necessarily use more pesticide and, for that matter what type of pesticide he used and what type of precautions he took when using pesticides or fertilizers.

Blair and Fraumeni (1978) conducted a study of the distribution of cancer of the prostate in some 3000 counties in the U.S.A. and showed a number of associations, one of which was a weak association between the number of chickens in the county and an increased risk of death from

cancer of the prostate. Other associations were observed for certain ethnic groups who were at higher risk. An association between high fat diets and prostate cancer was also reported.

Burmeister (1981) studied the age adjusted cancer rates in Iowa farmers dying from 1971 to 1978 and observed lower than expected mortality from tobacco related cancers of the lung, mouth and oesophagus and other respiratory cancers. Incidence of lip, stomach, leukemia, lymphatic, multiple myeloma and prostate cancer were higher in this group. The lower rates of lung cancer were associated with a lower use of tobacco by Iowa farmers (19 vs 44% of the population), (Pomrehn et al. 1982) but there was no clear evidence in this study of any excess cancer caused directly by pesticides because no direct information on the use of pesticides was available.

Burmeister et al, (1982) also conducted a further study of farmers in Iowa and noted an increased risk of dying from leukemia. The study was also based on analysis of death certificates of those persons dying during the years 1964-1978 and a total of 661 farmers were included in the study group. When various types of agricultural production were investigated, a higher incidence of leukemia was noted in counties where corn, soyabean and egg production were highest or where herbicide use was highest. Once again, this association was indirect, as no information on individual use of pesticides or farming operations was available from the death certificates.

Blair (1982) reviewed a number of studies on cancer risks associated with farming or rural living. Certain cancers were more common than would be expected and these were observed to include leukemia, lymphatic, prostate, skin, brain and stomach cancer. Morgan et al (1980) studied the cause of death in a large group (2620 individuals) of persons in the U.S.A. who were occupationally exposed to pesticides. Cancer of the internal organs was not significantly different from a control group but

pest control operators working in buildings and other structures did show increased frequency or incidence of skin cancer and dermatitis.

In a study on the incidence of skin cancer in England, Whitaker et al (1979) showed a higher than expected incidence in a number of professions including farming, however, pesticide use was not investigated and no conclusions could be drawn. In a survey of deaths from malignant melanoma between 1959 and 1967 in West Virginia, Scher et al (1981) showed a higher rate of incidence in agricultural areas than in urban areas. This increase seemed to be associated more with outdoor work and exposure to sunlight (a well known cause of skin cancer) than other factors. Stemhagen et al (1983) studied cases of primary liver cancer in New Jersey by death certificate information and interviews with relatives and associates of the deceased. Increased risks of dying of liver cancer were noted in all agricultural workers but information on specific pesticide or chemical exposure was vague and not usable in the analysis. Information on tobacco use in the agricultural group was not analyzed but the authors did suggest that its use may have been higher in the control group than in the liver cancer group. Increased lung cancer rates have been observed in sugarcane farmers in Louisiana (Rothschild and Mulvey, 1982) even when these are adjusted for tobacco consumption. Again, pesticide use could not be correlated precisely with cancer incidence and several other causes such as inhalation of fungal spores could also have been involved. Flanders et al (1984) studied the incidence of laryngeal cancer in Richmond County in Georgia and found that farmers showed higher rates of incidence than other professions. Pesticide use or exposure information was not available in the study.

In a Canadian study, Gallager et al (1983) investigated 84 multiple myeloma cases treated in Vancouver between 1972 and 1978 and showed an increased risk of this form of cancer in farmers. Data on the type of farming operation were not collected and no data were available on exposure to pesticides.

A study in Germany (Barthel, 1981) on a group of 1652 men in which 169 malignant lung tumors had been diagnosed showed that, in agricultural workers, the incidence of these tumors was twice that in the general population. In this case, it was possible to show that tobacco use in the two groups was not different and could therefore not account for the increase in lung cancer. The pesticides used in each case were not identified and other factors were not investigated as a possible cause of this phenomenon.

Graham et al (1977) showed in a study of testicular cancer from New York State that, amongst other factors, rural residence was associated with excess rates of this form of cancer. Mills et al (1984) studied the incidence of testicular cancer in the entire U.S. population and showed that this rare condition demonstrated a much higher risk odds in persons associated with agriculture. This type of testicular cancer is unusual in that it is more common in young men and is in fact the most common cancer in white males between the ages of 20 and 34. The fact that it occurs at this early age suggests that occupation is not an important factor in its cause. Neither of these studies addressed pesticides as a possible cause but Mills et al (1984) do suggest that some form of virus may be involved and that young men from rural and farm areas are exposed to the virus at a later and more vulnerable stage than their less isolated urban control group or that the virus is associated with animals and thus more likely to be found in association with farming as a profession.

Blair and Thomas (1979) studied the incidence of leukemia in Nebraska farmers on the basis of death certificate information from 1957 to 1974 and showed an increased incidence of this cancer in persons dying before 66 years of age and a significantly increased risk of dying of leukemia in farmers from counties where corn was in high production (and, in association, where hog and chicken production was also high). Although pesticide use in the production of corn is high, there was no increased

risk from leukemia in other counties where pesticide use was as high but on different crops.

In a study of a profession in which pesticides are also used, Blair et al (1983) used mortality data in licensed pesticide applicators in the state of Florida. In a group of 3827, of which 378 had died of known causes, incidence of lung cancer was higher than the control group and increased with increasing years of pesticide application. Brain cancer appeared to be higher than expected with 5 observed cases as opposed to the 2 which would be predicted from the control group. However, the total number in the case of brain cancer was low and may not reflect any real effect. The numbers in the case of lung cancer were more interesting as they did show a trend, however, no information on tobacco use was available and a similar trend would have been expected if smoking had increased during the period of employment. Wang and MacMahon (1979) carried out a cohort study of over 16000 professional pesticide applicators in the U.S.A. who had used pesticides for at least three months between 1967 and 1976. Excess deaths due to lung, skin and bladder cancer were seen in the applicators when compared to the general population, however, these were not statistically significant. Lower death rates were observed from cancer of the digestive organs as well as respiratory disease, however, these were also non-significant. Mancuso (1982) showed that brain tumors occurred less frequently in rural than urban men in Ohio in the years 1944-1952. No attempt was made to distinguish profession in the rural and urban males.

An interesting study (Wiklund, 1983) was carried out in Sweden on data obtained from the cancer-environment registry. In this study, the risks for most types of cancer (trachea, lung, bronchus, larynx, liver and tongue) were less in farmers than in the general population. The only cancer that showed a higher incidence was cancer of the lip which is thought to be associated with increased exposure to sun, wind and dust in a largely outdoor occupation such as farming. Burmeister and Morgan

(1982) also studied the incidence of excess mortality in Iowa farmers and farm laborers dying between 1971 and 1978 and showed that there was no increased incidence of death related to respiratory disease, although accidental death was higher than would be expected in this group. An earlier study in California (Carlson and Petersen, 1978) had shown an excess of respiratory disease (mainly pneumonia) in farm laborers as compared to farmers and farm managers.

A positive association between exposure to herbicides and mesothelial tumors of the ovary has been reported in a case-control study from Italy (Adalberto et al 1984). The report failed to identify the herbicides and the degree of exposure and, although case-controls were used, only age, year of diagnosis and area of residence were matched, leading to the possibility of confounding.

Other Health Effects in the Farming Community

Information on epidemiology of other possible health effects of pesticides on farmers was sparse. In a survey of persons in the U.S.A. exposed to pesticides as part of farming or pesticide application, Morgan et al (1980) showed an unusually high rate of accidental death, but rates of cancer and cardiovascular disease were similar to those of a control sample of unexposed individuals. Analyses of serum for organochlorine pesticide residues did show an association between levels in the serum and subsequent cardiovascular disease or hypertension, however, it is uncertain whether this is a cause and effect or a result of decreased ability to excrete the pesticide residues. A survey of members of the U.S. National Agricultural Aviation Association (Hopes Consulting Inc., 1980) compared the health of pesticide applicators exposed to pesticides as part of their occupation to that of unexposed brothers, sisters or spouses as controls. This survey did not show any increased incidence of miscarriages, stillbirths or birth deformities in those families in which one spouse (usually the husband) was occupationally exposed to pesticides.

Discussion

A potential problem in any study based on mortality or even health data is the comparison between data collected by two different persons. Data on profession of the general population is generally collected by a census and may, in the case of part-time farming or farm associated activities, be perceived differently than the data taken at time of death when this information would normally be taken by a health professional. Differences in this perception will introduce a bias into the ratio determinations which may be reflected in any analysis of the data, no matter how sophisticated the statistical techniques may be. Another confounding problem in such studies was suggested by Blair and White (1981) when it was observed that farmers in Wisconsin showed reduced tobacco usage as well as reduced tobacco related deaths such as lung cancer. Similar decreased rates of tobacco use and lung cancer were also observed in the agricultural profession in Finland by Pukkala et al (1983). The resultant bias in the study, where the control group was not similar to the observed group, may also bias the conclusions. A conclusion from the scientific study of the incidence of cancer in farmers is that in some cases, increased risks for certain cancers have been observed, however, there is no clear evidence that pesticide use is linked to this incidence of cancer. Good case-control studies which avoid some of the above problems could be carried out. However, most of those in the literature lack good controls or lack sufficient numbers to allow clear conclusions to be drawn.

Many of the epidemiological studies on cancer and farming have shown an association between this profession and higher risk for a number of types of cancer. Some of these may be expected such as those known to be associated with exposure to sunlight. Farming as a profession involves the use of many skills other than the application of pesticides and, in the role of welder, carpenter, mechanic and veterinarian, the farmer may be exposed to many other potentially carcinogenic

substances. In many cases, it seems as if these epidemiological studies were carried out with little science. No clear hypothesis was developed before the study and, as a result, little detailed information was collected on pesticides used, the use to which they were put, the type of application method, etc. A further problem is that no central source of information on the purchase or use of pesticides by individual farmers exists, either because the mechanism is not in place or because it is considered an invasion of privacy by most members of the public.

A prospective study of the relationship between cancer and pesticides would require accurate information on exposure to pesticides, information which not only includes the amount of pesticide purchased, but also the actual exposure in each individual farmer. Information on exposure to other hazards, tasks and chemical substances which are known to be carcinogenic would be needed as would be intensive, thorough and long-term medical followup of the farmers in question, a task which would be very costly. Given that the mechanism for the collection of this information is not in place and that the time difference between initiation and expression of cancer is often very long, such a study could only deliver results in the distant future. It is noted that there is a lack of epidemiological studies which address the effects of pesticide use on the health of farmers in Ontario.

CHAPTER SIX

FARM SAFETY MEASURES AND AWARENESS IN ONTARIO

The availability of farm safety literature in Ontario is good. Provincial Ministries and other organizations have published a number of booklets and other material (Table 6-1) relating to pesticide safety. In this wealth of information there is much duplication of effort and, to a certain extent, some inconsistency in the way information is presented and in the amount of information that is given.

Other sources of information on the safe use of pesticides are label information, extension persons, pesticide salespersons, agricultural representatives and dealers. It would thus appear that ample information on the safe use of pesticides is available to those who seek it. However, availability of information does not necessarily mean that it is used. A survey of farmers from Middlesex County carried out in 1979 (Bertens, 1979) suggested that awareness of the dangers of pesticides was present in the community but that most farmers were unaware of the need for, or availability of, information on safe use precautions. The survey was, however, poorly conceived, executed and reported and much other valuable and useful information was not reported. In a pesticide safety survey carried out in Alberta in 1983 (Hussain, 1983), a similar, but better documented situation was observed. Of 488 farmers interviewed, the following conclusions were drawn:

> About 10% of the farmers who used pesticides experienced what they believed to be symptoms of poisoning.
>
> Only one out of five supposedly poisoned farmers sought medical attention.

Of the supposedly poisoned farmers who had seen a doctor for treatment, only half of them were satisfied with the treatment provided. Nearly all the farmers said they were concerned about any health hazard that may be caused by pesticides and nearly half believed that pesticides were harmful to their health.

Although nearly 80% of the farmers said that they did their own pesticide spraying, only about one-third have received any instruction in the safe use of pesticides.

Severe symptoms of pesticide poisoning were very minor among farmers.

Only 3% of the farmers indicated that they experienced chronic health problems due to pesticide exposure.

Ten percent of the farmers said they were allergic to pesticides.

Although 65% of those farmers surveyed said that they wore some kind of special clothing and safety equipment when working with pesticides, very few wore the coveralls which are recommended as standard wear. The use of rubber gloves when needed was practised by only half the pesticide users. But it appeared that respirators and goggles were worn to the extent needed. Many farmers used a type of dust mask which was not recommended for use with pesticides.

Thirty-five percent of the farmers wore no extra protective clothing or equipment at all, either because they felt too uncomfortable, or for no given reason.

The use of a hard plastic cap was not practised by any farmer. The cap that was worn the most was made of a fabric which absorbed pesticides.

According to the survey, very few farmers seemed to recognize the presence of the hazard symbols on the pesticide label let alone associate any meaning to them.

It would appear from this survey that there is serious lack of awareness of the potential hazards associated with pesticides and other agricultural chemicals amongst farmers in Alberta. A survey of 256 farmers in Saskatchewan (Moore, 1982), showed similar trends, some of which are summarized below:

Almost 90% of farmers rinsed pesticide containers into sprayers but only 41% used the recommended three rinses.

About 66% of farmers did not dispose of containers in the recommended manner and 46% of farmers saved the containers for use on the farm.

Less than 10% of farmers used eye protection, less than 30% used respirators and the majority did not use "skin protection" (undefined in the survey) during the application of pesticides.

Between 25% and 50% of farmers felt that the information on pesticide safety supplied on the container was inadequate.

Farmers obtained information on the use and application of pesticides from the following sources:

Local dealer	75%
Chemical companies	66
Provincial Agriculture	42
Neighbours	36
Agriculture Canada	30
Farm management courses	28

Research stations	23
University	22
Government Agencies	9
Other	3

A study on this problem has recently been completed by the Ministry of the Environment (Verkley, 1984). Recommendations with regard to the safe handling of agricultural chemicals will depend to some extent on the actual situation in Ontario.

Discussion

While much information on safe handling of pesticides and other chemicals is available from a variety of sources, it appears that this information is not fully utilized. This may be due, in part, to inadequate presentation and distribution of safety information on pesticide products.

In addition, dealers and agricultural representatives supply much of the safety information on pesticides to the farm community. These persons may lack formal training in the hazards of pesticide use.

In a similar vein, the lack of adequate training and/or certification programs for farmers and agricultural workers in the hazards of pesticide use was noted.

Little information on the hazards associated with chemicals other than pesticides is available to Ontario farmers and farm workers.

Table 6-1

Publications Relating to Pesticide and Farm Chemical Safety

Title	Source
Using Crop Protection Chemicals Safely (booklet)	Canadian Agricultural Chemical Association
Safe Pesticide Usage and Disposal (audio visual)	Canadian Agricultural Chemical Association
Rinse for Dollars (poster)	Canadian Agricultural Chemical Association
Crop Protection Chemicals are Very Useful (pamphlet)	Canadian Agricultural Chemical Association
Pesticide Container Disposal (pamphlet)	Canadian Agricultural Chemical Association
Pesticide Container Disposal (poster)	Canadian Agricultural Chemical Association
1202-E Control of Fabric Pests	Agriculture Canada
1370-E Control of Rats and Mice	Agriculture Canada
1518-E Pesticides: Their Implications for Agriculture	Agriculture Canada
1543-E Pesticides for Home and Garden	Agriculture Canada
1736-E Insect Control in the Home	Agriculture Canada
1752-E Insect Control in the Home Garden	Agriculture Canada
5128-E Let's Talk about Pesticides	Agriculture Canada
Personal Protective Equipment for Pesticide Users	Ontario Ministry of the Environment
Chemical Safety Handbook	Ontario Ministry of the Environment
Pesticides Acts and Regulations	Ontario Ministry of the Environment
Chemical Storage Signs	Ontario Ministry of the Environment
Aluminum Phosphide for Groundhog Control	Ontario Ministry of the Environment
Captan Fungicide Guidelines for Use by Householders (pamphlet)	Ontario Ministry of the Environment
Various subjects under the title "Facts about Pesticides"	Ontario Ministry of the Environment
Handling Pesticides on the Farm	Farm Safety Association Inc.
The Perils of Pesticides	Farm Safety Association Inc.

Title	Source
Fact Sheet: Selecting the Correct Respiratory Protection	Farm Safety Association Inc.
Gas Poisoning on the Farm	Farm Safety Association Inc.
Anhydrous Ammonia	Farm Safety Association Inc.
Manure Gas	Farm Safety Association Inc.
Silo Gas	Farm Safety Association Inc.
Toxic Gas Warning Signs	Farm Safety Association Inc.
Farm Chemical Safety	Canada Safety Council
Respiratory Protection	National Safety Council
Danger Silo Gas	Canada Plan Service Agriculture Canada
Safe Use of Granular Pesticides	Ontario Ministry of Agriculture & Food
Pesticide Drift	Ontario Ministry of Agriculture & Food
Nitrates and Water Supplies	Ontario Ministry of Agriculture & Food
Protective Clothing When Using Pesticides Outdoors	Ontario Ministry of Agriculture & Food
Pesticide Container Rinsing	Ontario Ministry of Agriculture & Food
Pesticide Contamination of Farm Water Supplies	Ontario Ministry of Agriculture & Food
Sections of the following publications:	
Insect and Disease Control in the House Garden (pub. #64)	Ontario Ministry of Agriculture & Food
Tobacco Production Recommendation (pub. #298)	Ontario Ministry of Agriculture & Food
Fruit Production Recommendations (pub. #360)	Ontario Ministry of Agriculture & Food
Pesticide Recommendations for Greenhouse Ornamentals (pub. #381)	Ontario Ministry of Agriculture & Food

Title	Source
Guide to Chemical Weed Control (pub. #75)	Ontario Ministry of Agriculture & Food
Greenhouse Vegetable Production Recommendations (pub. #365)	Ontario Ministry of Agriculture & Food
Vegetable Production Recommendations (pub. #363)	Ontario Ministry of Agriculture & Food
Field Crop Recommendations (pub. #296)	Ontario Ministry of Agriculture & Food

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